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INFLAMMATION.

For the proper understanding of the descriptions of disease which I give in this work it is necessary that I state the views which I hold concerning inflammatory processes.

I believe that:

1. Inflammatory changes are morbid changes produced in the tissues by the action of irritants.

2. The morbid changes produced by the irritants are: degeneration, congestion, transudation, emigration, necrosis, and the growth of new tissue.

3. What we call inflammations are the occurrence of these morbid changes separately or together.

4. The symptoms of inflammation are due both to the poisonous properties of the irritant and to the morbid changes in the tissues; either one may predominate.

5. For clinical purposes the character of the morphological changes in the tissues is often of more importance than the character of the irritant.

I. The Irritants.

[illegible] The action of some of them seems [illegible] than this, to produce some [illegible] irritant is necessary. [illegible] bodies; irritating [illegible] poisons; dead [illegible] and cold; chronic [illegible] rheumatism; the [illegible] micro-organisms;

Pt. 1 - 1 Brain, [illegible]
Pt. 2 - 160 Lungs
" 3 320 Heart
" 4 400 Kidneys
" 5 535 Typhoid
" 6 605 Influenza, [illegible], [illegible], [illegible], [illegible]

LECTURES

ON

THE PRACTICE OF MEDICINE

WITH CASES AND CHARTS

BY

FRANCIS DELAFIELD, M.D., LL.D. (Yale)

PART I

INFLAMMATION
MENINGITIS
THE VENTRICLES OF THE BRAIN
THE CEREBRAL ARTERIES AND VEINS
THE PHARYNX AND TONSILS
THE LARYNX
THE PLEURA
BRONCHITIS

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I. The Irritants.

Of these there are many. The action of some of them seems plain, of others very obscure. More than this, to produce some inflammations the action of more than one irritant is necessary. The ordinary irritants are as follows: foreign bodies; irritating dusts; irritating gases; inorganic and organic poisons; dead tissue; malignant growths; the action of heat and cold; chronic congestion; the poisons of syphilis, gout, and rheumatism; the poisons of the infectious diseases; pathogenic micro-organisms; poisons of unknown nature produced in the body.

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Of these irritants, some act directly on the tissues: foreign bodies, irritating dusts, irritating gases, dead tissue, malignant new growths, pathogenic micro-organisms, inorganic and organic poisons.

Some are first introduced into the body by inoculation, inhalation, or swallowing, circulate in the blood, and are carried to the tissues which they are to irritate; inorganic and organic poisons: the poisons of syphilis, gout, rheumatism, and the infectious diseases; the toxins of the pathogenic micro-organisms; the unknown poisons produced in the body.

Some act on the blood vessels so as to produce local congestions: heat and cold and the mechanical causes of congestion, such as heart disease.

Not infrequently several irritants combine to produce inflammation. Streptococci, staphylococci, tubercle bacilli, diphtheria bacilli, are often found in varying combinations. A number of the different irritants may first cause congestion and exudation, and then a different character will be given to the inflammation by infection with pathogenic micro-organisms.

We know by experience something of the behavior of the different irritants.

1. A number of them regularly select some one part of the body, in which they set up inflammatory changes, without reference to the place or manner in which they are introduced into the body. Arsenic regularly causes gastritis; corrosive sublimate, colitis; the bacillus of cerebro-spinal meningitis, inflammation of the pia mater, etc.

2. The different irritants vary as to the particular form of the inflammatory processes which they are likely to produce. The toxins of some of the infectious diseases cause acute degeneration; the cholera bacillus causes large transudations of serum; streptococci produce emigration or necrosis.

3. The same irritant may produce different forms of inflammation. The tubercle bacillus may produce exudation, production of new tissue, or necrosis: the poison of scarlatina causes acute degeneration of the kidney, acute exudative nephritis, or productive nephritis.

4. The same anatomical form of inflammation can be pro-

duced by different irritants. The diphtheria bacilli and streptococci each produce a croupous inflammation of the throat; purulent inflammations are caused by streptococci or pneumococci; a productive nephritis can be caused by the poisons of scarlatina, of diphtheria, or of pregnancy.

5. The poisons of the infectious diseases do not always produce inflammations in proportion to their virulence, and they may even cause death without producing their characteristic inflammations.

6. In the present state of our knowledge we have to make a distinction between infectious diseases and infectious inflammations, although this distinction is often difficult.

Scarlet fever and rheumatism, for example, seem to be infectious diseases caused by a poison, with accessory inflammations which do not constitute the disease, but are only added to it. Diphtheria, on the other hand, is a local infectious inflammation, the patient poisoned by the toxins given off from the seat of infection.

7. Pathogenic micro-organisms seem to act as irritants in two ways. (*a*) The bodies of the bacteria, or the toxins produced by their growth, or both together, act directly as irritants on the tissues with which they are brought in contact and set up inflammatory processes.

(*b*) Some other irritant, such as cold or other atmospheric conditions, acts upon the tissues and causes congestion and exudation. Then this already inflamed tissue is infected by bacteria and the inflammation takes on the purulent or necrotic type.

The things also noticeable about micro-organisms are: that sometimes they do not act as irritants at all; that in different people and at different times there are great variations in their virulence either as local irritants or as producers of toxins; that most of the purulent and necrotic forms of inflammation are due to their presence; that in many inflammations the symptoms are due partly to the local changes, partly to the general poisoning caused by the toxins of the organisms growing in the inflamed tissue; that our knowledge is exceedingly imperfect as to the ways in which such organisms are introduced into the body, the detail of the formation of toxins, the apparent discrepancies between the number of demonstrable bacteria and the virulence of the general poisoning.

II. THE MORBID CHANGES PRODUCED IN THE TISSUES BY IRRITANTS.

These are: degeneration, necrosis, transudation, congestion, emigration, production.

Degeneration.

Definition.—I mean by this term changes in the substances which compose the cells of the viscera, the nerve fibres, and the muscular fibres. I do not include the so-called waxy, hyaline, fatty, glycogenic, mucous, colloid, and calcareous degenerations.

Etiology.—It is characteristic of such a degeneration as this that it does not occur by itself, but is always caused by the presence of some toxic substance. The poison may be an inorganic one, such as arsenic, phosphorus, or mercury; or an organic one, such as is produced by the growth of pathogenic micro-organisms. How the poisons are carried to the different parts of the body, and in what way the changes in the cells are effected, we do not know.

The poisons of the different infectious diseases vary as to the part of the body which they habitually select. In typhoid and typhus fevers the muscles are degenerated; in diphtheria the nerve fibres; in yellow fever the liver; in scarlatina the kidneys. In all the infectious diseases the renal epithelium is especially vulnerable and seldom escapes degeneration.

Morbid Anatomy.—The changes in the cells vary with the virulence and the quantity of the poison introduced into the body.

A mild poisoning only produces an increase in the size of the cells and a change in their composition, so that they are more opaque and more coarsely granular. The change in the cells is not necessarily attended with any disturbance of their functions. If the poison is withdrawn the cells return to their normal condition.

A more severe poisoning not only causes the cells to become swollen, but they are also infiltrated with granules and some of them disintegrate and break down.

In the more intense forms of poisoning there is a rapid death of the cells. They either disintegrate and break down, or pass into the condition of coagulation necrosis.

When the degeneration of the cells is marked and rapid there are often added congestion of the blood vessels, exudation of serum, and emigration of white blood cells. These changes in the blood vessels may be secondary to the degeneration of the cells, or they may be caused by the same poison that produces the degeneration of the cells; we have no exact knowledge.

The severe forms of degeneration cause disturbance or abolition of the functions of the cells of viscera. Degeneration of the nerve fibres produces motor paralysis and pain. Degeneration of the voluntary muscles may be followed by their rupture. The interference with the functions of the viscera and the paralysis may cause death. If the patients recover, the parts of the body in which the degeneration existed return to their normal condition.

Necrosis.

The ordinary causes of death of tissue are: a cutting off of the blood supply, severe concussions, and the growth of pathogenic bacteria. To the physician the necroses caused by the growth of bacteria are of especial importance.

The dead tissue is either changed into a mass of amorphous granules, or the cells pass into the condition of coagulation necrosis. The area of dead tissue may afterward shrink, be absorbed, and finally disappear; it may remain indefinitely in the condition of cheesy degeneration; it may soften, break down, and form a cavity; or it may undergo putrefaction.

Such a necrosis of tissue may occur as an isolated process without changes in the surrounding tissues. Much more frequently there are associated with it changes in the blood vessels and the tissues.

Transudation.

The serum of the blood may escape from the vessels as part of an inflammatory process; then we call it a transudation or exudation. It may escape under conditions which are not inflammatory; then we call it a dropsy.

The serum which is exuded in these ways is not always of the same character. Especially does it vary as to the quantity of albumin and of fibrin-producing substances.

All transudations of serum from the blood vessels seem to be produced in one of two ways:

1. The capillaries act as filters. The passage of blood serum through their walls follows the ordinary laws of filtration. Transudation is caused by increased blood pressure, by a change in the composition of the blood, or by changes in the walls of the capillaries.

2. The capillaries with their endothelium act as glands and secrete serum. Changes in the composition of the blood, irritative substances in the blood, or irritative substances in the tissues surrounding the capillaries can cause increased secretion.

Congestion.

The blood vessels, especially the capillaries and veins, in any part of the body may for a longer or shorter time contain an increased quantity of blood. This part of the body is then said to be in a state of chronic or active congestion.

Either active or chronic congestion interferes with the functions of the viscera.

Active congestion is often followed by exudative inflammation. Chronic congestion is often followed by productive inflammation.

Emigration.

The escape of the white blood cells from the capillaries and veins is usually associated with the transudation of blood serum, but may occur without it.

One cause for emigration is the presence in the tissues near the blood vessels of substances produced by bacteria which are positively chemotactic, that is, of substances which attract white blood cells toward them.

It is also found that a variety of irritating substances in the tissues are capable of causing an emigration of white cells. Apparently whenever the emigration of white blood cells is very large it is due to the presence of pathogenic bacteria, especially the streptococci and staphylococci.

The white blood cells which have emigrated into the tissues may remain for a time as pus cells and afterward degenerate and be absorbed. Furthermore, these cells are capable of taking into themselves bacteria and other foreign bodies, they can produce antitoxic substances, and it is probable that in this way they may be of use in limiting infection.

It is also to be noticed that when a local emigration of white blood cells is caused by bacteria there is at the same time an increase in the number of white cells in the blood throughout the body—leucocytosis.

Production.

With or without other inflammatory changes there may be a growth of new tissue. This tissue follows the general type of connective-tissue cells and basement substance. It is known by the names of granulation tissue, round-celled tissue, connective tissue, fibrous tissue, tubercle tissue, etc. As a rule the more acute the process the greater the number of cells; the more chronic the process the greater the quantity of basement substance.

In an acute inflammation the production of new tissue may occur by itself, but is more frequently associated with exudation and emigration. In chronic inflammations the growth of new tissue is often not attended with congestion, emigration, or exudation.

Whenever in any acute inflammation there is at the first a production of new tissue, that inflammation regularly goes on to assume the subacute or chronic form.

III. THE DIFFERENT FORMS OF INFLAMMATION.

To the phenomena produced by the irritants and by the morbid changes in the tissues caused by them we give the name of inflammation, and we classify the inflammations according to the character of the morbid changes in the tissues.

We distinguish, therefore:

1. Exudative inflammation.
   - *(a)* Simple exudative inflammation.
   - *(b)* Purulent exudative inflammation.

2. Productive inflammation.
   - *(a)* Simple acute productive inflammation.
   - *(b)* Acute productive inflammation with exudation.
   - *(c)* Chronic productive inflammation.

3. Necrotic inflammation.

1. *Exudative Inflammation.*

An exudative inflammation is one charactertized by the presence of an exudate—serum, fibrin, and pus. The production of such an exudation may or may not be attended with changes in the inflamed tissues. The process may run an acute, a subacute, or a chronic course. The character of the inflamed tissue, whether connective tissue, a mucous membrane, or a viscus, modifies the character of the inflammation.

We distinguish, therefore:

(a) Simple exudative inflammation.
(b) Exudative inflammation with large quantities of pus.

(*a*) So far as the exudate is concerned, we know by direct observation what it is and how it finds its way into the tissues.

In the inflamed tissue there is first a dilatation of the arteries, veins, and capillaries, and an increased rapidity of the circulation of the blood. Then the blood current becomes slower, while white blood cells accumulate in the veins and capillaries and adhere to the walls. This is the condition of "acute congestion," and the morbid process may not advance beyond this. If it does advance farther, the white blood cells change their shape, find their way between the endothelial cells of the blood vessels, through their walls, and appear on the outside of the vessels in the tissue. This is called "emigration." Red blood cells in smaller numbers may also pass through the walls of the capillaries and veins, and this is called "diapedesis." At the same time the plasma of the blood transudes through the walls of the vessels and infiltrates the tissues as serum; while, by the union of substances contained in the blood plasma and in the white blood cells, fibrin is formed.

In this manner are elaborated the inflammatory products—pus, serum, and fibrin. The pus cells are emigrated white blood cells; the serum is part of the plasma of the blood; the fibrin is produced by a union of the fibrinogen in solution in the blood plasma with substances contained in the white blood cells, and appears coagulated in the form of granules, amorphous masses, or a reticulum.

The relative quantity of pus, serum, or fibrin varies in different cases.

In simple exudative inflammation we find no other morbid changes than the congestion, exudation, and emigration; the tissues remain unaltered. When the inflammation has subsided all the parts return to their natural condition.

It is in connective tissue that a simple exudative inflammation is seen in its most typical form. The structure of connective tissue is simple—a basement substance, cells, blood vessels, lymphatics, and nerves. Inflammation is attended with congestion of the blood vessels, swelling of the basement substance and cells, the transudation of serum, which may or may not contain fibrin-producing substances, and the emigration of white blood cells.

The structure of the mucous membranes is more complex. They are all composed of a layer of epithelium, of a connective-tissue stroma containing the blood vessels, nerves, and lymphatics, and of glands which produce mucus. The inflammation not only causes the same congestion and exudation in the stroma, but there are also changes in the epithelium and the glands. In the epithelium there is a more active desquamation of old cells and growth of new cells; sometimes superficial ulcers are formed. The function of the mucous glands is interfered with. At first the production of mucus is stopped, later it is increased and altered. The increased production of mucus is regularly attended with a diminution of the congestion and swelling of the mucous membrane. Such an inflammation in a mucous membrane is often called "acute catarrhal inflammation."

The viscera are composed of a connective-tissue stroma containing the blood vessels, lymphatics, and nerves, and of cells. The cells are peculiar to each viscus and are concerned in performing the functions of the viscus.

The principal changes effected by the inflammation are the congestion and consequent swelling, the degeneration of the visceral cells and their inability to perform their proper functions. The quantity of exudation is small.

(*b*) In exudative inflammation with an excessive quantity of pus, or purulent inflammation, the excessive number of pus cells may or may not be accompanied by serum and fibrin. Such an excessive number of pus cells is due to an increased emigration of white blood cells.

The inflammation is of a more severe type than a simple exudative inflammation. The pus cells infiltrate the connective tissue, they are mixed with the serum in the serous cavities and with the mucus on the surfaces of the mucous membranes, but they do not form abscesses, but rather collections of pus.

2. *Productive Inflammation.*

(*a*) Simple acute productive inflammation. In this form there is no exudation, no serum, fibrin, or pus. Congestion is sometimes visible after death, but by no means always. The inflammatory product consists of new cells formed from the old connective-tissue cells. The pia mater and the peritoneum offer the best examples of this form of inflammation.

(*b*) Productive inflammation with exudation. In this form of inflammation the changes in the blood vessels, the exudation and emigration, the formation of serum, fibrin, and pus, are well marked, but in addition there is from the first a growth of new tissue. This new tissue at first consists principally of cells; later a basement substance and blood vessels are added. This form of inflammation has a marked disposition to continue for a long time in a subacute or chronic form.

In connective tissue the serum, fibrin, and pus are found in varying quantities. The new tissue forms thickenings and adhesions.

In the mucous membranes the inflammation involves the stroma, and it is in the stroma that the exudation is infiltrated and the new tissue formed. The glandular coat may remain unchanged or be the seat of catarrhal inflammation.

In the viscera the quantity of the inflammatory product varies. The new tissue is formed in the stroma. The visceral cells undergo more or less atrophy or degeneration.

(*c*) In chronic productive inflammation the inflammatory product is round-celled tissue, granulation tissue, or connective tissue. In some cases this is the only change; in others there is added an exudation from the blood vessels, or degeneration of cells. The new tissue that is formed may degenerate or become calcified.

In connective tissue this form of inflammation produces thickenings and adhesions, and serum in the serous cavities.

In mucous membranes the growth of new tissue is in the stroma. This is thickened, either diffusely or in the form of polypoid growths. The layer of epithelium may be thickened or thinned. The mucous glands are atrophied, or hypertrophied, or become cystic. The production of mucus is diminished, or increased, or altered. This condition in the mucous membranes is commonly called a "chronic catarrhal" inflammation.

In the viscera there is a growth of indifferent tissue or of connective tissue in the stroma. The visceral cells are compressed, or degenerated, or fatty, or disappear. The functions of the viscus are seriously interfered with. In the viscera this is often called an "interstitial" inflammation.

The most marked features of this form of inflammation are its slow course and its tendency to continue. The lesions of chronic productive inflammation, especially in old persons, are by some believed to be due to chronic degeneration.

3. *Necrotic Inflammation.*

In this form of inflammation, in addition to the congestion and exudation, there is added death of parts of the tissues in which the inflammation exists. This character of the inflammation is given to it by the presence and growth of pathogenic bacteria. The bacteria regularly present are the staphylococcus pyogenes aureus and albus and the streptococcus pyogenes. They frequently occur together in the same inflammatory process.

Such an inflammation, when it occurs in connective tissue, produces abscesses. A circumscribed portion of tissue is congested, infiltrated with serum, fibrin, and pus, and parts of the tissue die. The dead tissue softens, breaks down, and cavities are formed which contain serum, pus cells, and portions of dead tissue.

In mucous membranes there are different forms of necrotic inflammation. There may be circumscribed areas of necrosis involving only the glandular coat, due usually to streptococcus infection. There may be the so-called croupous or diphtheritic inflammations, with congestion, exudation of serum rich in fibrino-plastic substances, emigration of white blood cells, and necrosis of tissue either superficial or deep.

There may be deep necrosis due to infection with streptococci, amœbæ, or other micro-organisms.

THE DURATION OF INFLAMMATION.

It is a matter of practical importance that the inflammations, if left entirely to themselves, are governed by laws as to their duration.

The exudative inflammations are regularly transitory and of short duration. Most of them run their course within a week, few last longer than three weeks. If they are not fatal the tissues in which they have run their course return to their normal condition.

The combinations of exudative and productive inflammation behave differently. They run a longer course and are always liable to be followed by subacute and chronic inflammations. It is a pity that the characteristics of this form of inflammation are not generally understood. The ordinary assumption is that it is only a question of the severity of the inflammation. The mild forms are transitory, the severe forms are long continued. I feel sure, however, that the combinations of productive and exudative inflammation show their real character from the very outset. It is not a question of different severities of inflammation, but of different kinds of inflammation.

It is difficult to assign a definite duration to the necrotic inflammations; it depends largely on the extent of the necrosis and the virulence of the poisoning associated with them.

The Prognosis of Inflammation.

This depends on the character of the irritant, the extent and situation of the tissues inflamed, and the anatomical character of the inflammation.

The chemical irritants, the ptomaines, and the traumatic irritants vary according to their exact character and the severity with which they affect the tissues. If they only cause exudative inflammation they are not so bad, but if they cause necrosis they are serious.

The bacteria and their toxins are ordinary causes of inflammation. The perplexing thing about them is the varying virulence of the pneumococci, streptococci, tubercle bacilli, etc., both as to the severity of the local inflammation which they cause and the amount of general poisoning which accompanies the inflammation.

The extent of an inflammation is of much consequence. If the whole extent of the lungs, the whole length of the colon, or the whole surface of the skin is seriously inflamed, recovery is difficult.

Some parts of the body are especially vulnerable; even mild inflammations of the pia mater and peritoneum are easily fatal.

The anatomical characteristics of the inflammation, no matter how produced, are of great importance.

Necrotic inflammations are always serious and often fatal.

Productive and exudative inflammations are of severe type, of long duration, and are often followed by chronic inflammation.

The acute exudative inflammations are usually of mild type and short duration.

The chronic productive inflammations are the most curious of all. Without discoverable cause they exist for years in some individuals absolutely without symptoms, while in many others they are ordinary causes of death.

THE TREATMENT OF INFLAMMATION.

It is in the treatment of inflammation that we find the widest divergence between the point of view of the physician and that of the pathologist. The physician does not find it to the advantage of his patient to foster any of the phenomena of inflammation. Congestion, exudation, emigration, degeneration, necrosis, production of new tissue are all bad alike. His one object is to prevent or limit them.

Naturally the first object is prevention. If we can so arrange matters that none of the pathogenic bacteria can find entrance into the body, many inflammations and diseases can be avoided. Evidently the first step is to find out what the poisons are and how they are introduced into the body. Here we have made hopeful progress. There seems fair reason to hope that we will soon escape from the barbarous methods of quarantine and isolation to more rational methods of preventing the spread of poisons. On the other hand, it is to be remembered that the occurrence of some diseases in one generation after another diminishes malignancy and confers immunity.

Next come the attempts to prevent the growth of bacteria after they have been introduced into any part of the body. This affords a fascinating subject for study and experiment, but so far no practical results have been reached except in a few instances.

Equally important is the endeavor to neutralize the toxins of the pathogenic bacteria. This is a field in which much is to be hoped for, but as yet we have no real antitoxins except those for diphtheria and tetanus.

This leaves us with our endeavors to control degeneration, necrosis, production, congestion, exudation, and emigration. Experience teaches us that of all these changes the only ones we can control are those belonging to the blood vessels—congestion, exudation, and emigration.

In a certain number of cases we have real therapeutical means to use for this purpose. The local application of cold, heat, and astringents; the relief of tension by puncture and incision; the use of some elective drugs, such as aconite, salicylate of soda, ipecac, antimony, castor oil, etc.; the removal of venous congestion by sweating, catharsis, venesection, and mercury—all these are measures of which the effects can be daily seen, but they have their limitations.

Inflammations of the skin and superficial mucous membranes are controlled by heat, cold, and astringents.

Inflammations of the pharynx and tonsils are controlled by aconite and salicylate of soda; those of the larynx by antimony; those of the bronchi by ipecac and counter-irritation.

The exudative inflammations of the pia mater are apparently not affected by any therapeutic measures.

Pleurisy with effusion is cured by tapping; empyema is cured by opening the chest.

The different forms of inflammation of the parenchyma of the lung—the pneumonias—are not affected by any treatment.

For the inflammations of the stomach and colon we can do a great deal; about the small intestine we know nothing.

Abscesses of the liver can be cured by tapping and incision.

The acute inflammations of the kidney can often be controlled; with the chronic inflammations we can do little.

Some of the inflammations of the peritoneum can be cured by incision.

THE PIA MATER.

The external surface of the brain is overlaid by a connective-tissue membrane which covers the convolutions, dips down into the sulci, and extends into the ventricles. This membrane is abundantly supplied with blood vessels, and from it numerous vessels extend into the brain, so that any disturbance in the circulation of the blood in the pia mater involves a disturbance in the circulation in the brain also.

The connective tissue which makes up the pia mater is arranged in a series of membranes and fibres reinforced by elastic tissue, so disposed as to form a spongy membrane containing numerous cavities more or less filled with fluid. These cavities are continuous with the perivascular spaces which surround the vessels that pass from the pia mater into the brain. The outer layers of the pia mater are the most compact, and are covered on their outer surface by a continuous layer of endothelial cells. This external layer of the pia mater is often described as a separate membrane, called the "arachnoid," but it is really only part of the pia.

The deeper layers of the pia contain the blood vessels. The membranes and fibres which compose the pia mater are partly coated with cells, which have irregular and delicate cell bodies and large, distinct nuclei.

In all inflammations of the pia mater the inflammatory products regularly collect in the spaces within it. Only occasionally do we find them on its free surface.

The pia mater is frequently thickened, opaque, and white, either in diffuse patches or along the course of the vessels. In other cases single or multiple white spots, of the size of a pin's head or smaller, may be seen in the membrane, not appreciably elevated above the surface, but due to localized thickening. These slight opacities of the pia mater are commonly believed to be dependent upon repeated congestions of the membrane or upon chronic meningitis, but there is no evidence that this is always the case. They are most frequently found in old persons, but may exist at any age, and do not necessarily indicate the pre-existence of disease, although similar appearances are common in the chronic insane and in drunkards.

Congestion and Anæmia.—There can be little question that the quantity of blood contained in the vessels of the pia mater varies at different times, and that any considerable increase or diminution of the quantity of blood is likely to give cerebral symptoms. In the human subject, however, our knowledge of this subject is as yet indefinite.

Œdema.—The quantity of serum beneath the pia mater and in its cavities is very variable. It may accumulate as a result of atrophy of the brain substance or of venous hyperæmia, and may be accompanied by œdema of the brain substance also. It is not infrequent to find in hospital patients suffering from chronic nephritis, cardiac or pulmonary disease, or chronic alcoholism, a very considerable quantity of serum in this situation, and yet the patient has been free from cerebral symptoms.

ACUTE MENINGITIS.

Lesions.—The ordinary form of acute inflammation of the pia mater is the exudative, but occasionally the inflammation is of the pure productive type without exudation. Either form of inflammation, however, is attended with the same symptoms.

In acute productive or cellular meningitis the pia mater is congested, its surface is dry and lustreless, it is somewhat opaque, it is not at all infiltrated with serum. These changes in the gross appearance of the membrane are not marked and are easily overlooked, but the minute changes are more decided. There is an abundant production of cells somewhat resembling the cells which coat the surfaces of the membranes and fibres which make up the pia mater. This cell growth is general, involving the pia mater over most of the surface of the brain, but the cells are much more numerous in some places than in others. The inflammation, then, is one which results in the production, not of serum, fibrin, or pus, but of new connective-tissue cells, sometimes with a few pus cells. I have seen this form of meningitis with otitis media and occurring without discoverable cause. The duration of the cases was from six days to sixty days, so that it is evident that it is not a stage of ordinary meningitis, but a separate form. The cell growth was in some cases most marked at the base, in others over the convexity, in others over the whole brain. In the case which lasted sixty days there was also inflammation of the ependyma of the lateral ventricles.

In acute exudative meningitis there is an accumulation of serum, fibrin, and pus in the meshes of the pia mater and along the course of the vessels, rarely on the surface of the pia. Sometimes one, sometimes another of these exudations preponderates, giving rise to serous, fibrinous, or purulent forms of the inflammation. The absolute quantity of the exudation varies greatly. The quantity of exudation may be so small that the pia looks nearly normal to the naked eye, and the pus and fibrin can only be seen with the microscope. More frequently the quantity of exudation is considerable, and often very large, even sufficient to flatten the convolutions of the brain. The cortical portion of the brain may be œdematous, or degenerated, or infiltrated with minute hemorrhages. Very often the inflammation extends to the ventricles, which then contain purulent serum. In children the ventricular lesion is regularly well marked, the ventricles are dilated and contain large quantities of purulent serum. In adults such a distension of the ventricles occurs less frequently. The inflammation of the ventricles may persist for days and weeks after the subsidence of the meningitis.

The exudation may cover the whole surface of the brain, or be confined to the base or to the convexity. It often extends down on the pia mater of the cord, and the roots of the cranial nerve may also be involved.

Apparently in all forms of acute exudative meningitis micro-organisms are to be found in the exudation—streptococci and staphylococci, pneumococci, the diplococcus intracellularis, tubercle bacilli, or the bacilli of typhoid fever.

Causes.—Perhaps the best-marked form of meningitis is that which occurs in epidemics and is caused by the diplococcus intracellularis. Then come the cases secondary to inflammation of the middle ear with streptococcus or pneumococcus infection. Then come the cases which complicate pneumonia, influenza, rheumatism, endocarditis, and the other infectious diseases—mostly due to streptococcus or pneumococcus infection. Then come the cases due to infection with tubercle bacilli. Then the cases of infantile meningitis, and, lastly, the sporadic cases.

EPIDEMIC CEREBRO-SPINAL MENINGITIS.

The best account of this disease is the report of the State Board of Health of Massachusetts, 1898.

Definition.—An infectious disease of which the characteristic lesion is an acute inflammation of the pia mater of the brain and of the spinal cord.

Synonyms.—Spotted fever, malignant purpura, typhus petechialis, typhoid meningitis.

History.—The first unquestionable descriptions of the disease are those given by Vieusseux and Mathey of an epidemic which occurred in Geneva in 1805. Its distinctive features were an abrupt attack during the night, vomiting, severe headache, rigidity of the spine, difficult deglutition, convulsions, petechiæ, death in from twelve hours to five days. After death a gelatinous or puriform exudation was found on the surface of the brain and of the medulla oblongata. Between the years 1806 and 1816 there were localized epidemics in Germany, Holland, France, England, and the United States. Then there was an interval of six years during which the disease disappeared. In 1822-23 there were small epidemics in France and in Connecticut. Then, after another interval of five years, it reappeared in 1828 in Ohio, two years later in England, and three years later in Naples.

After four years of quiescence the disease entered upon a wider career, which was almost uninterrupted, from 1837 to 1850, and extended over a large part of Europe and of the United States.

Between 1850 and 1854 the disease again disappeared. Then there was a very fatal epidemic in Sweden which lasted until 1861. In 1856-7 there were epidemics at different places in the United States. From 1860 to 1865 the disease was seen in Holland, Portugal, and Germany. From 1861 to 1872 there were epidemics in many parts of the United States, the disease prevailing in New York in 1872 and 1874. In this country during 1898-1899 it prevailed in mild form in twenty-seven States. Sporadic cases are seen from time to time in places where the disease has been epidemic.

Etiology.—It is evident, from the history of the disease, that it is characteristic of it to appear and disappear during periods of years at places widely separated from each other. Its greatest prevalence and mortality have been in the northern portions of the temperate zone in Europe and America, and during the cool seasons of the year.

No locality seems to be exempt from the disease. It is seen in cities, villages, isolated houses in the country, barracks, prisons, and workhouses, without reference to soil or elevation. While all ages are liable to the disease, the greatest liability is among children and young adults.

It is generally believed that the poison of the disease is not transmitted from person to person, but is communicated to a number of persons at the same time.

The characteristic micro-organism of the disease is the diplococcus intracellularis meningitidis. The presence of this organism as the cause of the disease distinguishes this form of meningitis from the forms that are caused by pneumococci, streptococci, or tubercle bacilli.

The diplococci regularly find their way to the membranes of the brain and spinal cord and excite an acute inflammation. It is not known how these organisms are introduced into the body nor how they escape from it. They are found in the interior of the polynuclear leucocytes wherever there is an inflammation, but not in the blood or uninflamed tissues.

Lesions.—The characteristic lesion of the disease is an acute inflammation of the pia mater of the brain and spinal cord. In the great majority of cases this is a simple exudative inflammation, with an infiltration of the pia mater with serum, fibrin, and pus, these products accumulating in the largest quantity at the base of the brain and on the posterior surface of the cord. The lateral ventricles contain a little purulent serum. In children the quantity of purulent serum in the lateral ventricles is often so large as to distend them.

In a few cases there is no fibrin, serum, or pus, nothing but an increase of cells in the pia mater.

In a few cases it is said that the pia mater is congested, while no exudate is present.

The cortex of the brain may be œdematous, softened, and infiltrated with pus cells.

The inflammation may extend from the pia mater to the cranial and spinal nerves. The most marked lesions are usually found in the second, fifth, and eighth nerves. The exudation is between the sheath of the nerve and the nerve, or may infiltrate the nerve itself.

In the eyes there may be conjunctivitis or choroiditis.

In the ears there may be purulent inflammation.

In the lungs there may be a broncho-pneumonia, due to infection with the diplococci, or ordinary lobar pneumonia with a pneumococcus infection.

In the joints there may be simple or purulent synovitis.

In the blood there is regularly a large increase of the polynuclear leucocytes.

Symptoms.—The different epidemics of the disease have varied as to whether the invasion was sudden or preceded by a prodromic period.

If the prodromic period exists it is characterized by general malaise, headache, pains in the bones, loss of appetite, conjunctivitis, chilliness, and a slight rise of temperature.

When the invasion is sudden it is marked by one or more of the following symptoms: chills, a rise of temperature, headache, vomiting, tenderness and contraction of the muscles of the back of the neck, irregular pains in different parts of the body, conjunctivitis, restlessness, sleeplessness or delirium, and prostration.

After the disease is fairly established the following are the regular symptoms: Pain in the head—usually frontal, sometimes occipital or general, regularly severe and constant throughout the disease. The suffering is very great, so that the patients complain of it most bitterly, and even after they have become partially unconscious, from time to time they cry out with the pain. Exceptionally the pain is but moderate and intermittent.

Pain, tenderness, and contraction of the muscles of the back of the neck, or also of the muscles along the vertebral column, are often present. The head alone is drawn back, or the entire body is bent back and rigid. Any attempt to move the contracted muscles gives intense pain.

There may be severe pain along the course of the spinal nerves, pain which moves rapidly from one part of the body to another. With this pain there may also be hyperæsthesia of the skin. Later in the disease the pain and hyperæsthesia are succeeded by numbness and insensibility.

There may be contractions of groups of muscles, especially of the muscles of the face, or general convulsions; the latter are especially common in children.

Instead of involuntary contraction of the muscles, there may be paralysis of the muscles of deglutition, or of those of one of the extremities, or of those of a large part of the body. Ptosis, strabismus, and paralysis of the bladder and rectum occur quite frequently.

The eyes are liable to a variety of inflammations—conjunctivitis, keratitis, or choroiditis. Independently of inflammation there is at first photophobia with contracted pupils, later insensibility to light with dilated pupils.

The hearing is at first unnaturally acute and painful; later it is dulled or lost. In some epidemics a large number of the patients suffer from inflammations of the ear, which leave behind permanent deafness.

Restlessness, sleeplessness, delirium, stupor, and coma are present in various degrees, either early or later in the disease, or throughout the entire course. The delirium is mild, or active, or so violent as to resemble acute mania. The establishment of well-marked delirium and stupor alternating with each other, one or the other predominating as the disease goes on, belongs to the severe cases.

Vomiting is a very common symptom at the beginning of the disease. It may be so frequent and distressing as to constitute for a time the most important symptom.

The bowels are usually constipated, but during the later stages of the severe forms of the disease there may be diarrhœa and involuntary evacuations.

In some epidemics it is reported that the joints were inflamed. Sometimes they are swollen, red, and tender; or red and painful without any swelling; or swollen and painful without redness. The joints usually attacked are the knee, elbow, wrist, and the small joints of the fingers and toes.

The characteristic pulse of the disease is one which is slow in proportion to the height of the temperature. Pulses of 60 and 70 to the minute are quite common, and ones even as low as 27 are recorded. But exceptions to this rule are not infrequent, and in the severe cases the pulse regularly becomes more rapid as the disease goes on.

In the accounts of the earlier epidemics of the disease, when no thermometer was used, it is frequently stated that there was no fever. Since the introduction of the thermometer it has been found that there is a rise of temperature, often as high as 104° F. But the curve of temperature is very irregular and not in correspondence with the progress of the disease. A patient may be doing as badly with a temperature of 99° F. as with one of 104° F.

Eruptions of the skin are present in some epidemics, absent in others. They have been observed more frequently in the epidemics in America than in those in Europe. When the disease first appeared in New England a large proportion of the cases exhibited petechial eruptions and ecchymotic spots, whence the disease received the name of spotted fever. There have been observed in different epidemics ecchymoses in the skin, petechiæ, erythema, roseola, urticaria, and pemphigus.

The severity and mortality of the disease have varied in the different epidemics. In some cases the disease has been of most malignant type, the mortality as high as 75 per cent. and many of the cases dying within a few hours. In others the mortality has not been over 20 per cent and the duration of the disease has been from one to four weeks.

In children the invasion of the disease is often marked by convulsions, which may afterward be frequently repeated. The temperature is high and the pulse rapid throughout the disease. During most of the time the children are in a condition of stupor alternating with restlessness.

In the regular cases there may be a temporary cessation of the symptoms for several hours, or even for two or three days, and they then go on as before.

The disease may be complicated by bronchitis, pneumonia, pleurisy, pericarditis, or endocarditis.

In the last epidemic of this disease in New York, in 1872-74, all the cases began with a sudden invasion or chill, fever, vomiting, severe headache, and delirium. The clinical picture was always that of a severe meningitis with marked cerebral symptoms. The large majority of the cases were in persons under thirty years of age.

The treatment employed at that time consisted of cold applications to the head and the administration of quinine, potassium bromide, chloral hydrate, and morphine. This treatment seemed to have no effect on the mortality, which was considerable.

CASE I. *Epidemic Cerebro-Spinal Meningitis; Death.*—Male, 23 years old, March 10, 1872. Yesterday morning he was suddenly seized with a severe chill and intense pain in the frontal region; in the evening he vomited a number of times and felt very sick. To-day the man looks very ill and complains of the severe frontal headache, no pain or stiffness of the back of the neck, no physical signs over the heart or lungs. Urine 1025, a trace of albumin.

Third day: A profuse epistaxis, some photophobia, the tongue is dry and coated.

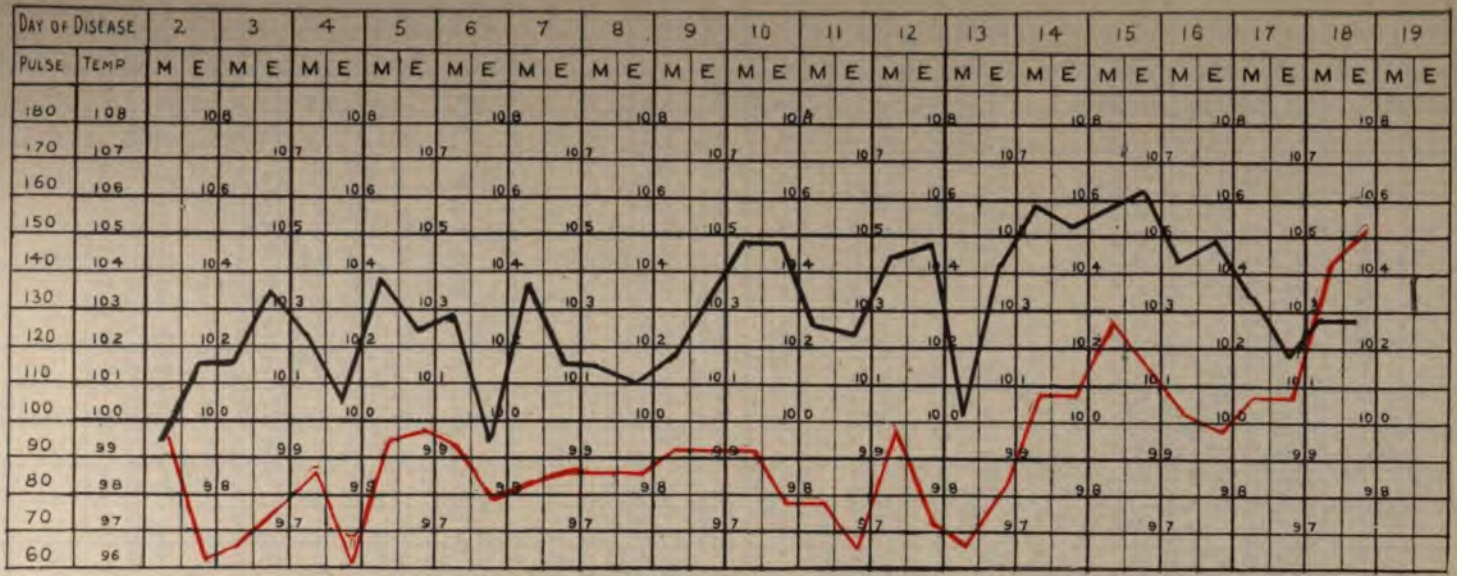
Fourth day: Pain and tenderness in the muscles of the back of the neck.

Eighth day: Patient feels better; he slept all night, and has but little headache to-day.

Fifteenth day: The man has been fairly comfortable with good nights until to-day. Now he is worse. The eyes are suffused, he is restless and delirious, there is general hyperæsthesia, the tongue is dry, but there is no headache.

Eighteenth day: The delirium continues with constant moaning, the face is emaciated and ghastly, the eyes half open, the

FIGURE 1.



Temperature black; pulse red.

head is drawn back, there are coarse râles over both lungs, the evacuations are involuntary. In the evening he died.

(For chart of Case I, see Fig. 1.)

Autopsy.—The pia mater over the whole surface of the brain is infiltrated with purulent serum. The pia mater of the cord is congested, but there is no exudation to the naked eye. There is purulent serum in the right pleural cavity. The upper part of the lower lobe of the right lung and of the upper lobe of the left lung are consolidated.

The liver is fatty.

The kidneys are congested.

CASE II. *Epidemic Cerebro-Spinal Meningitis; Recovery.*—Male, 17 years old, March 12, 1872. Yesterday morning he was suddenly attacked with severe pain in the head, active delirium, and vomiting. To-day he is violently delirious, the pupils are dilated, the head is drawn back; the urine is retained, specific gravity 1021, a trace of albumin.

Seventh day: The violent delirium and sleeplessness are subsiding. He is much quieter and sleeps for several hours at a time; the tongue is not so dry.

Ninth day: The patient is rational, sleeps well, has some appetite, but has troublesome pains in the arms and legs, and the right elbow joint is hot and swollen.

Twentieth day: He has been steadily improving, but there is a small abscess about the middle of the left thigh, and the glands in the left groin are enlarged.

Twenty-sixth day: The man is up and about the ward.

(For chart of Case II, see Fig. 2.)

CASE III. *Epidemic Cerebro-Spinal Meningitis; Death.*—Male, 35 years old, April 2, 1872. Two days ago, in the evening, he began to have pains in the hips, back, neck, and back of the head, and the head was drawn backward.

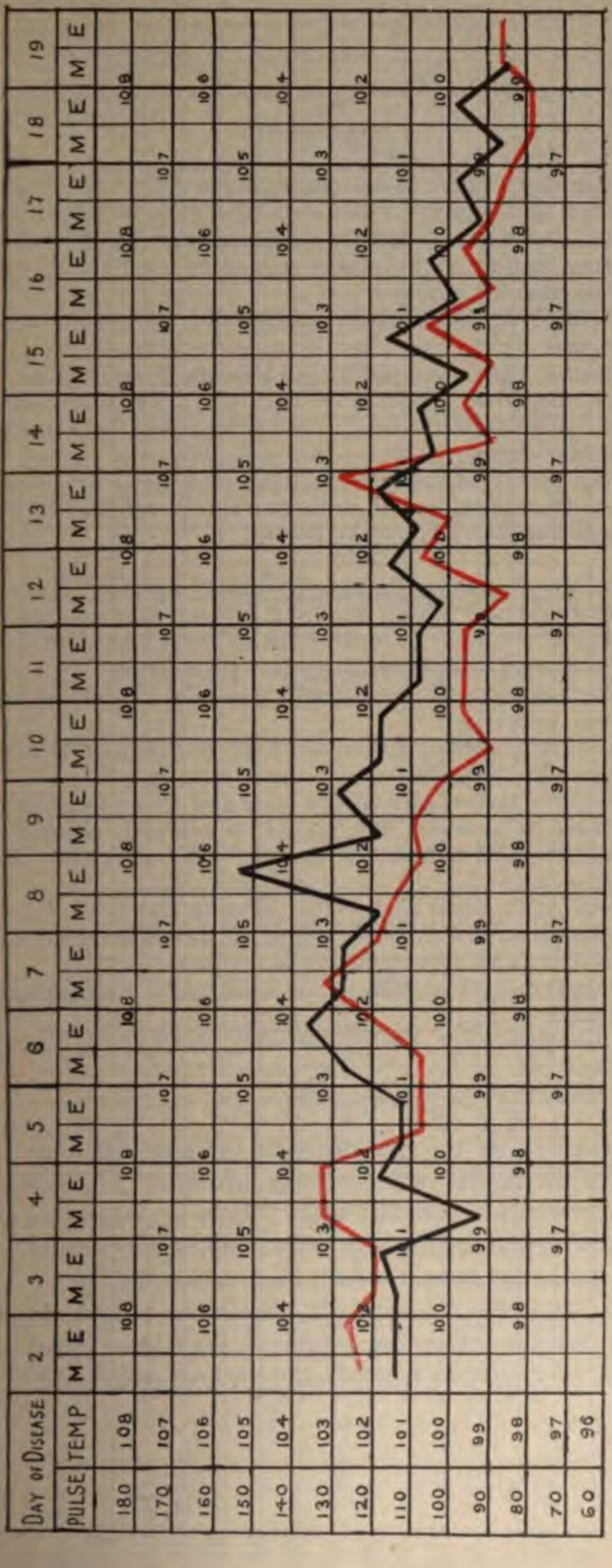
The next day he was about the same, no chill, no vomiting, no photophobia, and in the afternoon the headache ceased.

Third day: Pulse 72-72, temperature 102°-101.5° F., no eruption. He is perfectly sensible, complains of pain in the neck and back; the head is much drawn back.

Fifth day: Pulse 68, temperature 100.3° F. He is mildly delirious and restless, but can answer questions rationally. He says he has no pain except in the back of the neck; the tongue is coated and dry.

Sixth day: Pulse 68-72, temperature 100.3-100.5° F. The patient continued restless and sleepless in the day and became stupid in the evening. The pupils are contracted.

FIGURE 2.



Temperature black; pulse red.

Seventh day: Pulse 84-96, temperature 100.3°-100.7° F. The man is now quite stupid; there are coarse râles over both lungs.

Eighth day: Pulse 120-126, temperature 100.3°-102.7° F.; emaciating rapidly; alternating stupor and delirium; urine retained; would not swallow.

Ninth day: Pulse 126, temperature 103° F.; in the afternoon quietly died.

Autopsy.—The entire pia mater is intensely congested; at the base it is moderately infiltrated with pus; the lateral ventricles contain a little turbid serum. The substance of the brain is intensely congested.

The pia mater of the cord and the cord itself are congested; the pia is thickly infiltrated with pus all the way down the cord. The lungs and kidneys are congested.

CASE IV. *Epidemic Cerebro-Spinal Meningitis; Recovery.*—Male, 18 years old, March 25, 1872. Four days ago was seized with chill, fever, pain in head, back of neck, and shoulders; the head was drawn backward; there was photophobia.

The next day he felt better and was able to walk about with very little pain.

Third day: Violent pains in the head and back, with vomiting, diarrhœa, and epistaxis.

Fifth day: Severe pain in the head and neck; vomiting; head drawn back; tongue dry and coated; is rational; general hyperæsthesia. In the afternoon became delirious, with constant moaning.

Twelfth day: Until to-day the pains in the head, back, legs, and abdomen have continued, and there has been some delirium every day, but to-day everything is better.

Twentieth day: The pain in the head and delirium came back for two days.

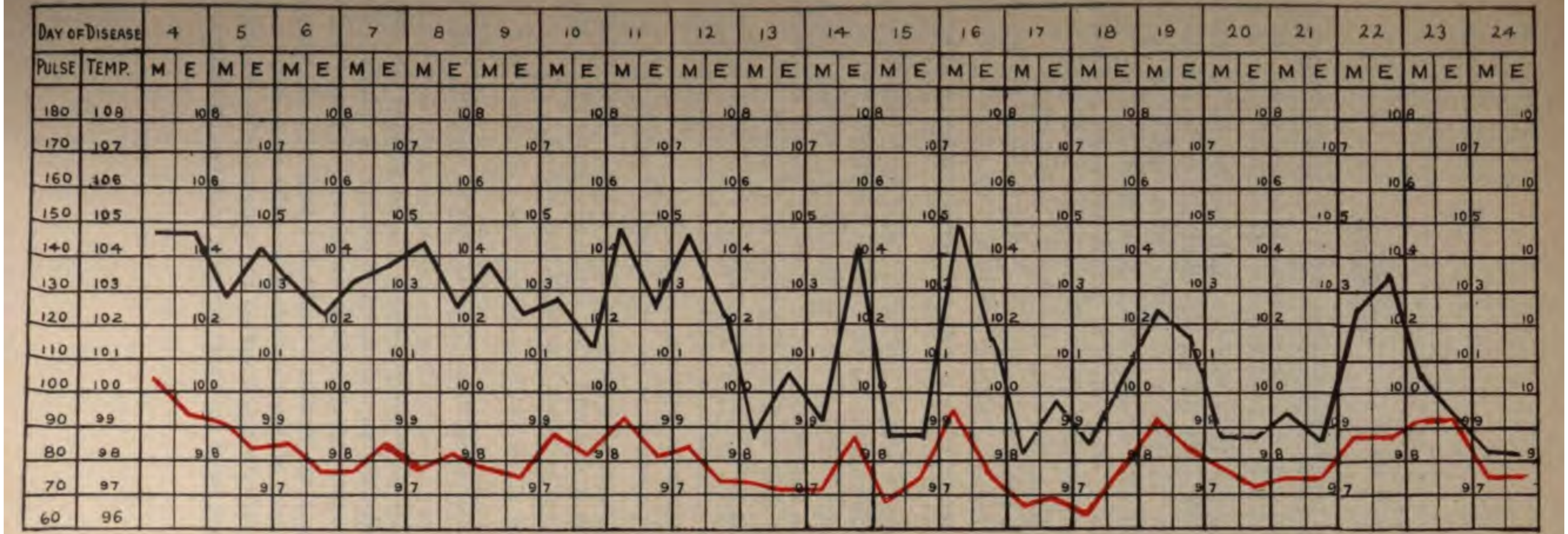
Twenty-eighth day: He has been doing well and is now up and about the ward.

(For chart of Case IV, see Fig. 3.)

Sequelæ.—After the subsidence of the meningitis the inflammation of the lateral ventricles may continue. The patients begin to improve, but soon fall back. There is progressive emaciation, an irregular febrile movement, alternating stupor and delirium, and, after several weeks, the patients die.

Other sequelæ are: blindness, deafness, mental feebleness, aphasia, anæmia, hysterical symptoms, disordered menstruation, paralysis of groups of muscles, and chronic endocarditis.

FIGURE 3.



Temperature black; pulse red.

The prognosis of the disease varies in different epidemics, regularly worse during the commencement and height of each epidemic.

Treatment.—There are two main indications for treatment—the meningitis and the general condition of the patient.

For the meningitis we apply continuous cold to the head and administer repeated small doses of calomel or the sulphate of magnesia. The iodide of potash and ergot are also used for the same purpose. These measures are most likely to be efficacious during the early days of the meningitis.

For the headache, restlessness, sleeplessness, and delirium we give the bromides, chloral hydrate, or opium.

If the temperature is high cold sponging and cold tubbing are used.

The heart's action may be so feeble as to require the use of alcoholic stimulants, digitalis, or strophanthus.

Excessive vomiting can be relieved by feeding the patients with small quantities of milk to which oxalate of cerium, bicarbonate of soda, or sulphonal have been added.

The bowels should be kept open. It must be admitted that so far we seem to have no treatment which really controls either the meningitis or the toxic poisoning.

At the present time the withdrawal of spinal fluid by lumbar puncture is used a good deal for purposes of diagnosis and treatment, both in this and other forms of meningitis. I cannot say that I like it or think it to be of real service.

MENINGITIS SECONDARY TO INFLAMMATIONS OF THE EAR.

Acute purulent inflammation of the middle ear occurs frequently in children and is not uncommon in adults. The cases vary as to their severity, their duration, and their liability to be complicated by meningitis. In the majority of cases there is inflammation of the mastoid cells. The rule seems to be that there is no meningitis unless the inflammation of the mastoid is purulent, but I have seen one case with fatal meningitis and an autopsy in which the mastoid was only congested and infiltrated with serum.

In children the pain in the ear and head is severe, the temperature runs high, there may be delirium, general convulsions, and the appearance of serious illness, so that they look much as if they had meningitis. But inside of a week the membrana tympani is perforated, the pus is discharged, and recovery is rapid. If, however, a secondary meningitis is developed the cerebral symptoms and the high temperature continue.

In adults the pain in the head is not as severe; the temperatures are not high; there are no cerebral symptoms; the meningitis may not come on until after the otitis has lasted for several days.

In some cases there will be found the ear full of pus, the mastoid infiltrated with pus, circumscribed patches of pus and fibrin on the corresponding surfaces of the dura mater and pia mater. This looks as if the inflammation was directly transmitted from the ear to the pia mater.

In other cases with the same ear lesions the dura mater is not inflamed, the meningitis is diffuse, and it seems as if the inflammation might be due to general septic infection rather than to direct transmission.

In the majority of cases thrombi are found in the sinuses of the dura mater near the inflamed ear.

The infection is regularly with streptococci or pneumococci.

The inflammation may be localized in the pia mater over the temporal bone, or spread over the base, or involve the whole of the pia. There may be a sufficient quantity of serum, fibrin, and pus to be evident to the naked eye. But in adults quite frequently the pia looks normal or congested. No fibrin or serum is present. There is nothing but an infiltration of the pia with new connective-tissue cells and perhaps a few leucocytes.

As a rule, both in children and adults the meningitis gives marked and regular symptoms: pain in the head, photophobia, strabismus, delirium, stupor, muscular contractions, general convulsions. The urine often contains albumin and casts. The temperatures in children run up to 104° or 105° F., in adults often not over 102° F.

Case V.—Male, 19 years old, September 16. Six weeks ago he was taken ill with pain in the right ear and right side of neck, no chill, no headache. He was in bed for two weeks, then thought he was well for two days; then had chills, fever, and sweating at irregular intervals. His mind was somewhat confused, his bowels were loose.

First day: Pulse 96-100, temperature 102°-103.5° F. The tongue is coated and dry; the urine is normal; there is pain in the right ear and the right knee; he is a little jaundiced.

Second day: Pulse 112-125, temperature 102°-105° F.; he is delirious.

Third day: Pulse 132-128, temperature 102°-105° F.; the delirium is violent.

Fourth day, he died.

Autopsy.—Over the lower surface of the right lobe of the cerebrum the pia mater and the superficial layer of the brain are infiltrated with pus over an area one inch in diameter. Corresponding to this on the outer side of the dura mater is a thick collection of pus; beneath this the bone is discolored. There are soft, dirty thrombi in the sinuses of the dura mater.

The middle ear is full of pus.

The heart is normal.

The lungs contain a number of hemorrhagic infarctions.

The liver and spleen are large and soft. The kidneys are large and succulent.

Case VI.—Female, 18 years old, February 27. Two weeks ago she had a sore throat for three days. This was followed by pain in the right ear. This ear soon broke and discharged freely. Four days ago she had pain in the left ear and the left side of the head. She has vomited, her bowels have been loose, and she has had pains and tenderness over the abdomen, and the feet are œdematous.

Fifth day of meningitis: The skin is very pale, the face anxious, the pupils normal, the tongue is dry and coated. She complains of very severe headache; is rational, but answers slowly and looks very ill. The urine contains a good deal of albumin and many hyaline and granular casts.

Sixth day: Pulse 128-112, temperature 103.4°-101.2° F.

Seventh day: Pulse 124-120, temperature 103°-101° F.; headache, dry tongue, hyperæsthesia of the skin.

Eighth day: Pulse 104-136, temperature 98°-102° F.

Ninth day; Pulse 144-160, temperature 101°-102.2° F. She is very restless, with slight convulsive movements of the muscles of the legs.

Tenth day: Pulse 152-152, temperature 102°-101.6° F. She is delirious.

Eleventh day: Pulse 150-144, temperature 103°-103.4° F. She was stupid all day and died quietly in the evening.

Autopsy.—The right middle ear and the cancellous portion of the right temporal bone are full of pus.

The right lateral sinus, where it passes through the posterior lacerated foramen, contains a firm thrombus.

The left ear and temporal bone are normal.

The pia mater is opaque; it is everywhere infiltrated with polygonal cells and leucocytes, no fibrin or serum.

In the heart there is a small fringe of vegetation on the mitral valve.

The lungs are congested.

The kidneys show the changes of acute degeneration.

If the meningitis follows a chronic otitis there will be the history of the chronic ear trouble extending back for months or years; then suddenly come on the symptoms of the meningitis—fever, headache, alternating delirium and stupor, muscular twitchings, coma.

Treatment.—The preventive treatment consists in the free incision and scraping of the mastoid cells. This seems to be good practice, but in children it should not be done unless there is really purulent infiltration of the mastoid. After the meningitis is once established the operation on the mastoid does not seem to be of much service.

We endeavor to control the inflammation by the local use of cold, and treat symptoms. But it must be confessed that such a meningitis usually goes steadily on and terminates fatally inside of two weeks.

MENINGITIS COMPLICATING THE INFECTIOUS DISEASES.

The poisons of all the infectious diseases are capable of causing meningitis. It is seen most frequently, however, with pneumonia, malignant endocarditis, surgical sepsis, typhoid and typhus fevers.

All of these infectious diseases are capable of giving marked cerebral symptoms without any inflammation of the pia mater.

When a complicating meningitis really is developed, the only difference is that the cerebral symptoms are more marked and approach more closely to those of an idiopathic meningitis.

A meningitis with pneumonia seems to be part of a general pneumococcus infection, and we are apt to find purulent pleurisy and pericarditis in the same person.

Case VII.—Forty-five years old; had always been well and strong until nineteen days before her death; then, while washing windows, became cold and wet and felt unwell for several days. Nine days before her death she had chills, pains in the back and bones, fever, sharp pain beneath the right nipple. Four days before death she gave the physical signs of consolidation of the upper lobe of the right lung. The temperature ran between 103° and 104° F., the pulse between 100 and 140. There was no sputum. She developed a muttering delirium, contractions of the muscles, became weaker, and died nine days after the initial chill.

Autopsy.—There was a diffuse infiltration of purulent serum in the pia mater. There was a little purulent serum in the pericardial sac.

Both pleural cavities contained purulent fibrin and serum. The upper lobe of the right lung was hepatized.

With malignant endocarditis meningitis is found not infrequently, but it can hardly be said to give definite symptoms. The cerebral symptoms belonging to the endocarditis itself are often so marked that the diagnosis is uncertain. As all the patients die, this is not a matter of much consequence.

Case VIII.—A man, 43 years old, nine days before his death complained of general malaise and vertigo. During the next two days there were repeated rigors and vomiting. Four days before death he became delirious; tongue brown and dry; small ecchymoses in the skin; moist râles over both lungs. The temperature ran from 102° to 103° F., the pulse from 116 to 150. He died comatose after an illness of nine days' duration.

Autopsy.—Beneath the pia mater of the convexity there was a thin layer of fluid blood. The pia was infiltrated with groups of polygonal cells and leucocytes and a little fibrin. The capillaries contained an unusual number of leucocytes.

There were ecchymoses in the pericardium.

The aortic valves were thickened, calcified, and stenosed; the ventricular surfaces were covered by soft vegetations beneath which were ulcers.

There were ecchymoses in the pulmonary pleura; the bronchi were congested and contained mucus and pus.

The spleen was large, soft, and contained recent infarctions.

The kidneys were large, contained old and recent infarctions, and great numbers of small red and white foci.

When meningitis is caused by sepsis after an injury or a surgical operation, the cases vary as to whether the symptoms of the sepsis or of the meningitis predominate.

Case IX.—Male, 30 years old, eleven weeks before death fell through a coal hole and struck his perineum. Ever since that time he has had difficulty in passing water. Six days before death an external urethrotomy was done. The same evening he had two chills. The next day his temperature was 100° F.; he was feeble and drowsy. The following day his temperature was 99° to 102° F.; he complained of pains all over the body, alternating restlessness and delirium; pupils contracted. The day before death the temperature was 105° F.; he had two general convulsions, followed by partial paralysis of the right side; the right knee joint was swollen. He died with a temperature of 107° F. six days after the operation.

Autopsy.—Over the entire surface of the brain there was pus in and beneath the pia mater.

The lungs were congested and the pulmonary pleura was coated with fibrin. There were no thrombi in the veins. The wound and the tissues around it looked healthy, except for an accumulation of pus about the prostatic urethra.

Tubercular Meningitis.

Tubercular inflammation of the pia mater behaves differently in children and in adults, so that it is necessary to describe the disease separately according to the age of the patient.

1. *Tubercular Meningitis in Children.*

Lesions.—The dura mater is usually unchanged, but there may be miliary tubercles on its inner surface. The surface of the brain is flattened by the pressure of the fluid which distends the ventricles. The inflammatory process in the pia is a combination of tubercular and of exudative or cellular inflammation, either one of which may predominate. The tubercles are very small and transparent, or large and white. They are composed of simple aggregations of small cells or of well-formed tubercle tissue arranged around blood vessels. The vessels show the changes of obliterating endarteritis. The cellular inflammation produces new connective-tissue cells; the exudative inflammation produces serum, fibrin, and pus. The inflammation is often confined to the base of the brain, but may extend over its entire surface or be confined to the convexity. The tubercles are most numerous in the pia mater over, and in, the sulci.

The ventricles of the brain are much dilated and distended with serum. Their ependyma is often thickened and studded with very small miliary tubercles. There may be miliary tubercles or larger tubercular nodules in the brain tissue. Miliary tubercles are usually present in the other serous membranes and the viscera. The kidneys are in the condition of acute degeneration or acute exudative nephritis.

The tubercles in the ependyma of the lateral ventricles are so small that they are not noticeable. But if vertical sections of the ependyma are made it will be found that they are present in the majority of cases. In fact, in a large number of the cases of tubercular meningitis of children the lesions of the lateral ventricles are more important than those of the pia mater.

In some cases miliary tubercles and fibrin, serum, and leucocytes are also present in the pia mater of the cord.

Such a tubercular meningitis is nearly always only part of a general tuberculosis, so that we regularly find miliary tubercles in the other serous membranes and the viscera.

Causes.—The children affected are usually under five years of age. There is often a family history of tubercular disease. The meningitis is regularly only one of the lesions belonging to an acute general tuberculosis. Such a general tuberculosis may be a primary disease or secondary to a localized tuberculosis.

The most common primary focus is in the bronchial glands. These are the seat of tubercular inflammation usually secondary to a broncho-pneumonia.

Symptoms.—It is important to remember that the tubercular meningitis is usually only part of a general tuberculosis, and that the intensity of the inflammation of the pia mater varies much in different cases. So we find that in some cases the symptoms are more like those of a general tuberculosis, in others like those of a meningitis, and that the meningitis may have the characters of an acute or a subacute inflammation.

There may be a prodromic period, probably due to the development of the general tuberculosis, which usually lasts for only a few days, but may be protracted for several weeks. During this period there are loss of flesh and strength, slight evening fever, irritability, sleeplessness, frontal headache, vomiting, constipation, and a coated tongue. These symptoms are not continuous, but are interrupted by periods of improvement.

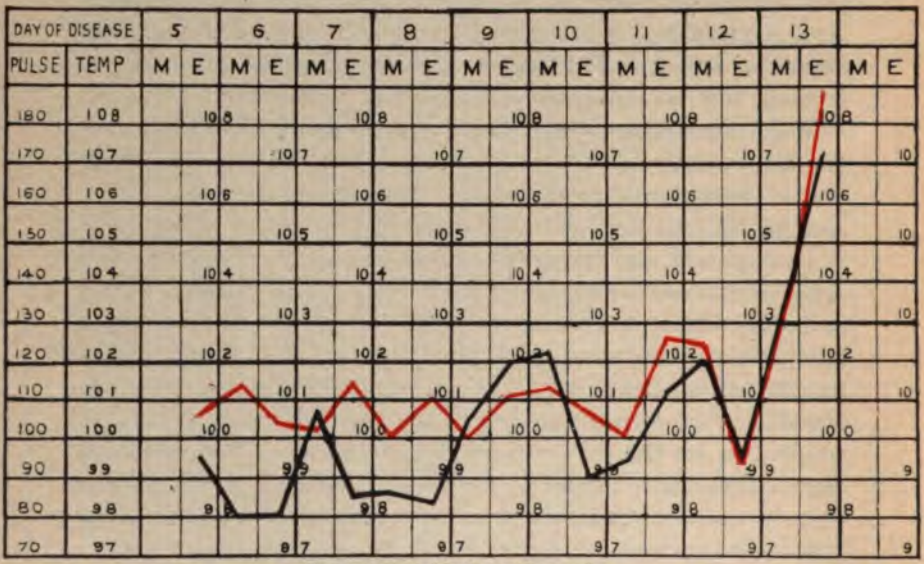
When the disease is established the child lies in bed, the face flushed, in a condition of alternating stupor and delirium, either one of which may predominate. During the periods of restlessness the child seems to have severe pain in the head; there is photophobia and hyperæsthesia of the skin. There is often frequent and active vomiting. The child rolls its head, moans and cries out; it may be actively delirious; there may be one or more general convulsions. During the period of stupor it lies quietly in bed, taking no notice, but apparently placid and comfortable. The bowels are constipated. The pulse is slow in proportion to the temperature, but irregular. The breathing also is irregular. There is a febrile movement which runs a very irregular course, but is not necessarily at any time very high. The urine is scanty and contains albumin.

At the end of the first week, or a little earlier or later, strabismus, inequality of the pupils, and ptosis are developed.

In the second week the stupor deepens into coma, which may be continuous or alternate with active delirium. The head is drawn back, there are convulsive movements or paralyses or rigidity of the limbs and face, which may be transient or permanent. There may be automatic movements of the arms and legs. The patients constantly pick at the nose and lips. The pupils are dilated and vision is lost. The pulse becomes very rapid and feeble. The course of the disease is interrupted by short periods of improvement; it lasts for from two to four weeks. The patients die in an attack of convulsions, or they become very feeble and emaciated; the urine is suppressed, the pulse and breathing are very rapid, the temperature remains high or falls below the normal. This is the history of the cases with general tuberculosis and a moderate meningitis.

Case X.—Boy, 2 years old. Three months before death he had pneumonia, and since that time has not been strong. Thirteen days before death he began to vomit. The vomiting has continued at regular intervals, without reference to the ingestion of food. He has not cared to eat; the bowels have moved but seldom; he has been alternately fretful and crying, or listless and quiet. He was in this condition for four days. On the fifth day the vomiting was frequently repeated, the apathy deepening into stupor varied with restlessness and crying.

Figure 4.



Temperature black; pulse red.

On the eleventh day there were a number of general convulsions; he refused food; there was marked stupor.

On the twelfth day the convulsions were repeated; there were projectile vomiting, automatic movements of one leg. The blood showed marked polynuclear leucocytosis. He died on the thirteenth day of the disease, the convulsions continuing nearly up to death.

(For Chart of Case X, see Fig. 4.)

Autopsy.—The pia mater of the brain and spinal cord is everywhere congested and studded with miliary tubercles. The lateral ventricles are dilated, distended with serum, and the brain tissue around them is softened.

The bronchial glands are enlarged by tubercular inflammation. The lungs, liver, spleen, and mesenteric glands are studded with miliary tubercles.

It is possible, however, for such a meningitis to be more intense and prove fatal within a week. The disease then usually begins with repeated convulsions followed by a stupor. The temperature is high and the pulse rapid.

On the other hand, some cases run a long course of several weeks' duration, with intervals of considerable improvement. In the following case the symptoms extended over fourteen weeks.

CASE XI.—Boy, 2 years old, admitted to the hospital on December 6; weighed eighteen pounds four ounces. Seven weeks before admission he began to vomit and has vomited frequently ever since.

On admission the temperature is 99.8° F., pulse 148, breathing 36. The urine is of a specific gravity 1020, contains a little albumin and a few hyaline and granular casts.

After admission the vomiting was less frequent and he began to take food.

Fifty-second day: Lumbar puncture, one ounce of turbid fluid.

Fifty-sixth day: Quiet, hungry; weighs nineteen pounds three ounces.

Sixtieth day: Pupils dilated; irritable; vomiting.

Sixty-second day: Stupor; vomiting; pulse irregular; lumbar puncture, one ounce of turbid fluid; weighs nineteen pounds.

Sixty-fourth day: Involuntary contractions of muscles of arms and legs.

Sixty-sixth day: Lumbar puncture, six drachms of turbid fluid.

Seventy-second day: Weighs eighteen pounds two ounces; lumbar puncture, one drachm of fluid; stupor increasing.

Seventy-sixth day: Weighs fifteen pounds four ounces; stupor alternating with fits of crying. Automatic movements of eyes and jaws.

Eighty-second day: Hungry and thirsty; lumbar punctur one drachm of clear fluid.

The child continued to emaciate with alternating stupor an crying, nystagmus, involuntary contractions of the muscles face, rigidity of the muscles of the extremities and of the nec loss of vision, and finally died after an illness of fourteen week

(For chart of Case XI, see Fig. 5.)

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Case XII.—Female, 6 years old, August 6, 1872. The chi was taken sick two weeks ago with vomiting, headache, and feve The eyes were congested; the bowels moved infrequently; s became dull and stupid.

On the fifteenth day she was stupid; the abdomen retracte rigidity of the muscles of the back of the neck, with tendernes pupils dilated; hyperæsthesia of the skin; slight opisthotono temperature 100.5° F., pulse 130, breathing 26. She remaine in the same condition until the twenty-first day, when she had convulsion and died.

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There was a layer of fibrin over the posterior surface of th spinal cord for its entire length.

There were miliary tubercles in the pleura, lungs, liver, per toneum, and spleen.

Treatment.—The treatment varies with the acuteness of th meningitis. In cases which run an active course, like that o an acute meningitis, we employ the same methods of treatmen as for that disease. In the cases which behave more like genera tuberculosis with subacute cerebral symptoms, the nursing, feeding, and alleviation of symptoms constitute the entire treatment

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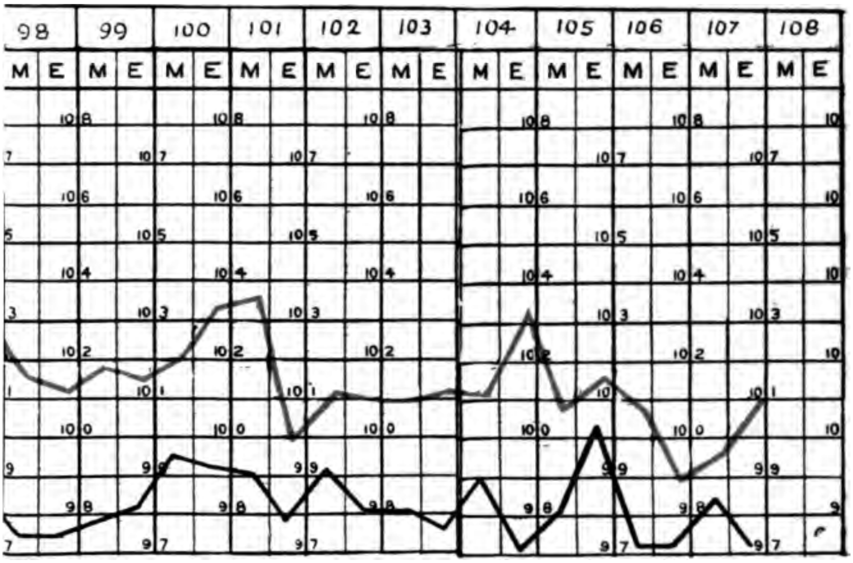
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Autopsy.—The pia mater of the brain was congested and at the base was infiltrated with fibrin. At the base and in the longitudinal fissure the pia was studded with large numbers of gray miliary tubercles.

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2. *Tubercular Meningitis in Adults.*

The changes in the pia—the combination of tubercular, exudative, and cellular inflammation—are the same as in children. There is, however, one important difference between adults and children. In children the inflammation of the ependyma of the ventricles, the distension of the ventricles with serum, and the consequent compression of the brain are the rule, while in adults they are not as frequent.

In adults, as in children, the tubercular meningitis may only be a part of a general tuberculosis. But more frequently it occurs as a localized tubercular inflammation, either primary, or secondary to a localized tubercular inflammation of some other part of the body.

Causes.—The greatest liability to the disease seems to be between the ages of fifteen and twenty-five years. Persons who inherit the tubercular disposition, or who have chronic pulmonary phthisis, are especially liable to the disease. It may very well happen that the pulmonary lesion is small and insignificant.

Symptoms.—It is convenient, in describing the symptoms of tubercular meningitis in adults, to divide the cases into five clinical groups:

1. Cases of acute general tuberculosis with tubercular meningitis, but without the symptoms of meningitis. The history of these patients belongs to general tuberculosis. The cerebral symptoms are only such as are seen in any severe infectious disease.

Case XIII.—Male, 34 years old, August 9. In March he began to feel weak and good for nothing, but without any definite symptoms. Two weeks ago he felt worn, with pains in the back and legs, no appetite, general feebleness, occasional nausea and vomiting.

August 9: The man was feeble, tremulous, no appetite, with anxious face, coated tongue, no physical signs, and the general look of a man who had been drinking hard. He continued in the same condition until August 16, when a mild delirium supervened and he looked as if he had typhoid fever. The tongue became brown and dry; the urine and fæces were passed involuntarily; there were twitchings of the muscles; he lay quietly in bed, taking no notice of anything, and died on August 30.

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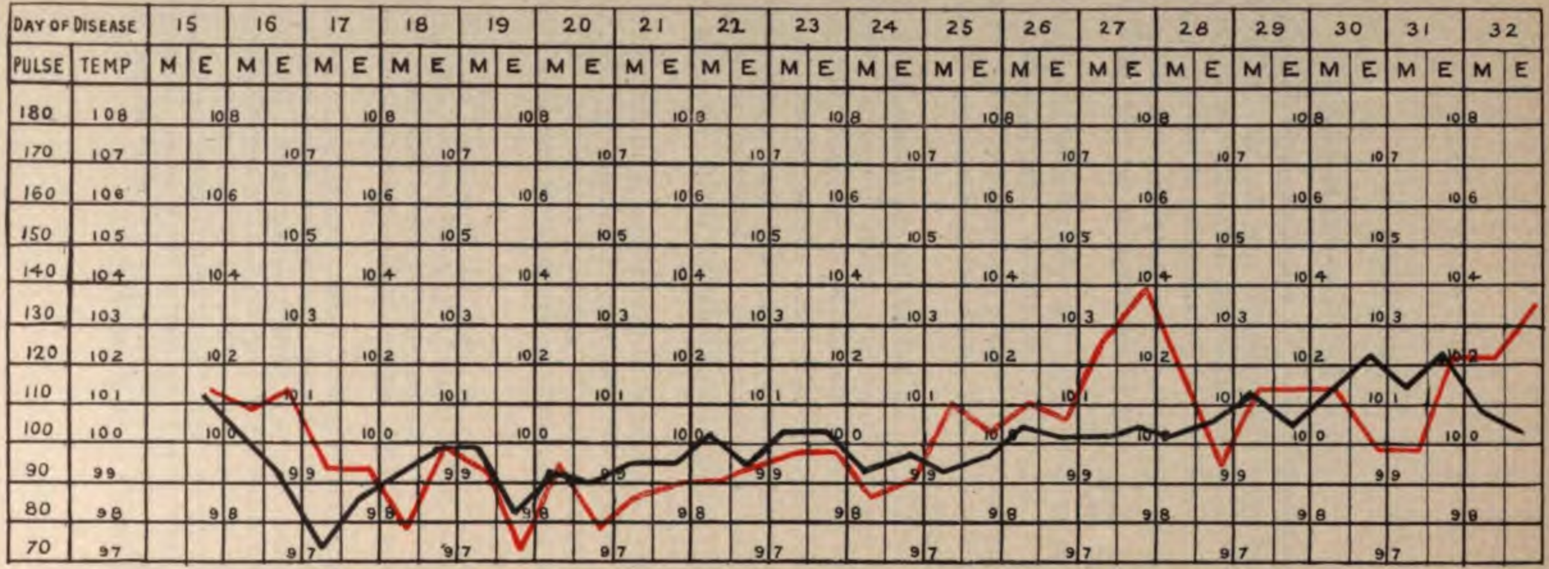


Temperature black; pulse red.

Autopsy.—The pia mater of the brain was dry and lustreless. At the base and in the fissures and sulci were small white tubercles.

There were miliary tubercles in the lungs, liver, kidneys, and peritoneum. There was an old tubercular focus in the left kidney.

2. Cases of general tuberculosis with tubercular meningitis with well-marked symptoms of meningitis. The disease behaves in these patients very much as it does in children. The cerebral symptoms are well marked and the cases vary principally as to their duration.

Case XIV.—Male, 42 years old. One week ago he began to have a slight cough and frontal headache. Five days ago he began to be delirious. On the eighth day he was feeble and emaciated and had delirium alternating with stupor, automatic movements; pupils normal; pulse 40, temperature 97.5° F. He died comatose on the tenth day.

Autopsy.—The pia mater over the base of the brain was infiltrated with fibrin and studded with miliary tubercles. There were miliary tubercles in the lungs and in the kidneys. No old tubercular focus could be found.

Case XV.—Male, 12 years old, January 24. Three months ago he had measles complicated with broncho-pneumonia, and was ill for six weeks. Since that time his health has not been good. Five days ago he became much worse. He had chills, severe headache with pains throughout the body, vomiting.

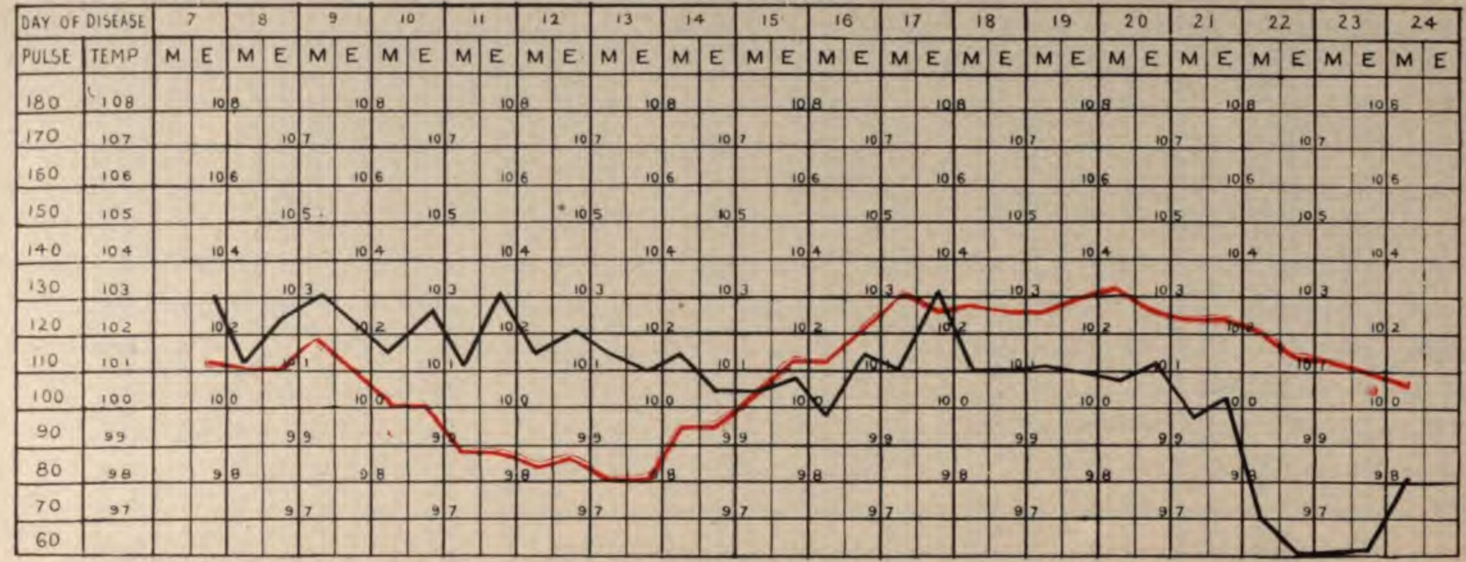
On the sixth day of the meningitis the patient was badly nourished, mind confused and dull, severe headache, no physical signs over the chest; urine 1030, no albumin, a few hyaline casts. The red blood cells were 5,712,000, the white blood cells 7,200, the hemoglobin 100 per cent. The headache continued to be very severe.

On the twelfth day restlessness, delirium, and muscular tremor alternated with periods of stupor. On the twenty-first day he became comatose and remained so until he died, on the twenty-fourth day of his illness.

(For chart of Case XV, see Fig. 7.)

Autopsy.—The pia mater all over the brain was studded with miliary tubercles; the pia at the base was infiltrated with fibrin; the lateral ventricles were moderately distended. There were miliary tubercles in the lungs, liver, pleura, and kidneys.

FIGURE 7.



Temperature black; pulse red.

3. Cases of tubercular meningitis without tubercular inflammation of the other parts of the body and without characteristic cerebral symptoms. These cases are obscure to the last degree. It is evident that the patient is suffering from a severe infectious disease, but the diagnosis is not usually made without an autopsy.

Case XVI.—Female, 24 years old, September 1. No history of any previous illness. Eight days ago began to feel tired, with headache and pains in the bones.

On the fourth day, in the evening, a chill; began to cough, with profuse, white sputum.

There was a little bleeding from the nose and some abdominal pain.

On the fifth day she took a cathartic and since then has had diarrhœa.

On the ninth day: The woman is still well nourished, the tongue is moist and coated; she has no appetite and a moderate diarrhœa. There were no physical signs over the chest. It was supposed that she had typhoid fever.

On the eleventh day: She began to have an active noisy delirium, and this continued up to the time of her death on the twenty-first day. There were no other symptoms, except the gradual loss of flesh and strength. The urine was normal.

(For chart of Case XVI, see Fig. 8.)

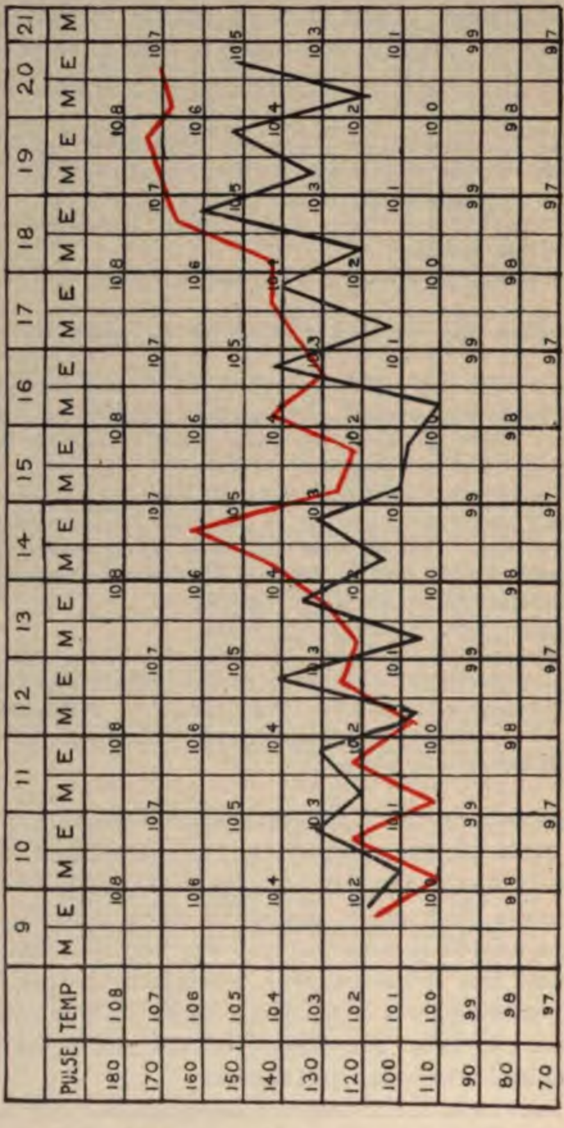
Autopsy.—Three hours after death.

The pia mater of the brain was whitish colored, but shining; there were a few small white miliary tubercles at the base of the brain. The lateral ventricles were distended with clear serum. There were no miliary tubercles in any other part of the body and no old tubercular focus.

4. Cases with well-marked symptoms of meningitis, which come on more or less suddenly, and are developed in persons whose health was apparently good up to the time of the attack. Of these cases there are a number in which a localized tuberculosis has existed in some part of the body, but has given so little trouble that it has not been recognized, so that when the secondary meningitis is developed it seems to be a primary inflammation. In other cases, however, the meningitis is a primary, localized tubercular inflammation.

In all these cases the invasion of the symptoms may be sudden or gradual. If it is sudden there are chill, fever, headache, vomiting, and so much prostration that the patient is at once

FIGURE 8.



Temperature black ; pulse red.

confined to bed. If it is gradual the patients do not feel sick enough to go to bed for several days. They have a little fever, headache, loss of appetite, and general malaise.

When the disease is fairly established the headache is severe and continuous. The patients are sleepless, restless, and go on to mild or active delirium. The delirium alternates with stupor or coma. There are conjunctivitis, photophobia, ptosis, strabismus, involuntary contractions of the muscles of the face, arms, and legs. From two to five days before death there may be permanent hemiplegia, monoplegia, or facial paralysis. Hyperæsthesia of the skin is present in some cases. Vomiting is often persistent and troublesome. A few days before death there may be difficulty in swallowing. In some cases there is pain and contraction in the muscles. The urine contains albumin and casts. The temperature may run low or high, and follows a very irregular course. Generally speaking, the cases of short duration have high temperatures, and those of long duration low temperatures. The pulse is irregular, sometimes slow and sometimes rapid.

The symptoms may be intense, continuous, and the patients die in from seven to fourteen days; or they may be less severe, with intervals of improvement, and the patients may go on for from thirty to fifty days.

Case XVII.—Male, 31 years old, August 29. His friends say that he has been sick for two weeks with vomiting, pain and stiffness in the back of the neck, and epistaxis.

Now he is in the condition of delirium alternating with stupor; does not answer questions; has great difficulty in swallowing; the urine is retained; pulse 48 and full, temperature 101° F. He continued in the same state of delirium and stupor, the temperature between 101° and 103° F., the pulse gradually increasing in frequency until it reached 156. He died on the twentieth day of the disease.

Autopsy.—The pia mater over the convexity of the brain is dry and lustreless, over the base it is infiltrated with serum and fibrin. There are miliary tubercles in the pia, in the fissure of Sylvius and the longitudinal fissure.

In the apices of the lungs there are small areas of black fibrinous tissue and hard, gray miliary tubercles.

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CASE XVIII.—Female, 17 years old, August 31. Her mother and two maternal aunts died of phthisis.

Her own health was good until three weeks ago. Then she began to have chills, fever, headache, prostration, and pains all over the body.

Now she is fairly nourished, color good, tongue moist and coated, urine 1015, no albumin. No physical signs over the chest or abdomen.

The headache, not very severe, continued until the close of the disease; she lay quietly in bed, gradually losing flesh and strength. On the forty-sixth day she had a convulsion. On the fiftieth day another convulsion. On the fifty-fifth day she passed fairly into the condition of stupor and lost control of the bladder and rectum, and on the sixty-second day she died.

(For chart of Case XVIII, see Fig. 9.)

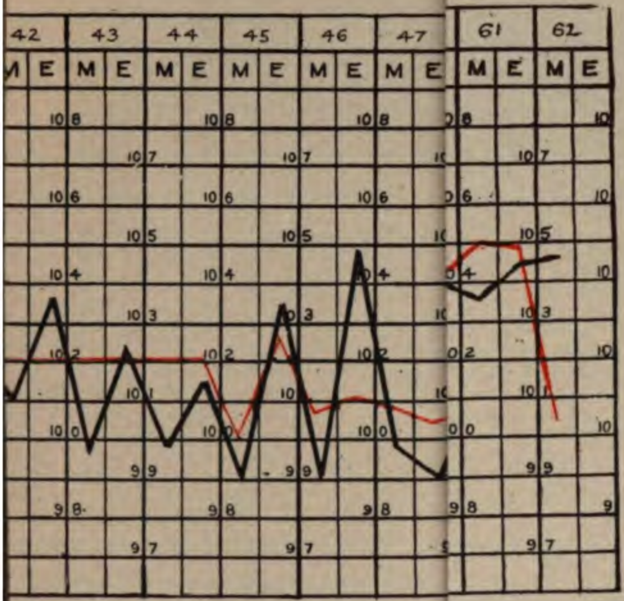
Autopsy.—There are a few white miliary tubercles in the pia mater of the sulci and over the convexity of the brain. At the base the pia is infiltrated with fibrin and pus. There is a recent broncho-pneumonia of the lower lobe of the right lung. There are no miliary tubercles in any other part of the body, and there is no old tubercular focus.

5. Cases with a well-marked history of pulmonary tuberculosis may at some time develop a tubercular meningitis. Such a meningitis regularly causes death, but yet does not give very characteristic cerebral symptoms.

CASE XIX.—Male, 33 years old, December 18. He says that he was well until three months ago. Then he began to have cough, muco-purulent sputum, several small hæmoptyses, afternoon fever, and loss of flesh and strength. Now he looks like a case of advanced phthisis, very much emaciated, with cough and muco-purulent sputum, tongue dry and brown, mild delirium. On January 2 he died.

Autopsy.—The entire pia mater is thickened and opaque, with miliary tubercles at the base of the brain. The upper lobes of both lungs are converted into fibrous tissue, honeycombed with large cavities. The lower lobes are studded with miliary tubercles and consolidated with red hepatization.

There are large tubercular ulcers in the small intestine.



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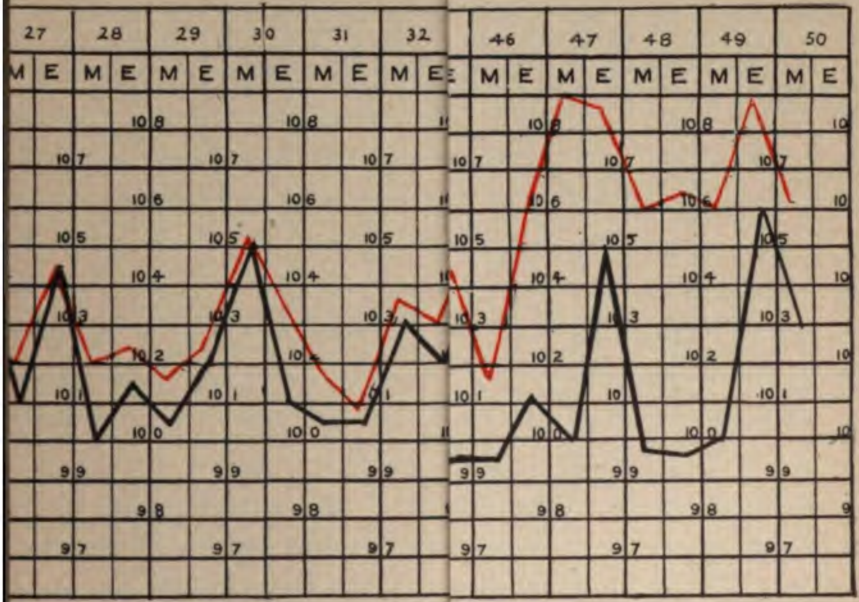

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SPORADIC CASES OF MENINGITIS IN NEW YORK.

Since the epidemic of cerebro-spinal meningitis in New York in 1872-73, I have seen every year cases of meningitis which were apparently examples of the sporadic form of the epidemic disease, although the bacteriological diagnosis was made only in a few of the cases.

Of these cases some began with an acute invasion and went on with well-marked cerebral symptoms for from seven to fifty days. The diagnosis in these cases was usually plain. Other cases, however, began with a slow and indefinite invasion; the cerebral symptoms were not at any time characteristic; the disease would last for from four to fourteen weeks; the diagnosis was often very difficult.

1. *The Acute Cases.*—In most of these cases the exudate in the pia mater of the brain is scanty—often not visible without the microscope. In some cases there is only a growth of polygonal cells (cellular meningitis), in others there is in addition a scanty infiltration with leucocytes and fibrin. The pia mater of the cord is little or not at all changed, as a rule, but occasionally there is a considerable infiltration of pus and fibrin.

The invasion of the symptoms is with vomiting, headache, body pains, and marked prostration. The patients go on with headache, photophobia, hyperæsthesia of the skin, contractions of the muscles, alternating delirium and stupor, and progressive emaciation. In some there is tenderness and rigidity of the back of the neck. The white blood cells are regularly increased in number. The symptoms may continue steadily up to the time of the patient's death, or there may be intervals of improvement. The temperature and pulse are very irregular, as will be seen in the charts of the cases. The disease is very fatal.

CASE XX.—A woman, 16 years old, April, 1883. Seven days before death she was suddenly attacked with chills, nausea, vomiting, headache, deafness, marked prostration, and a rise of temperature. The woman became steadily worse, the pulse from 100 to 104, the temperature from 101°-102° F., the deafness continuing with alternating delirium and stupor until she died.

Autopsy.—Six hours after death the pia mater of the brain was dry and lustreless and showed no exudate to the naked eye.

Minute examination showed an infiltration of the pia over the whole brain with leucocytes and polygonal cells.

There were no lesions of the ears or of the temporal bones. In the lungs there was marked general congestion and bronchitis; the rest of the body was normal.

Case XXI.—Male, 11 years old, June, 1900. The boy had always been well, had received no injury, nor had any ear symptoms.

Three days before admission to the hospital he complained of malaise and fever and vomited. On the second day he passed into the condition of alternating delirium and stupor, with rigidity of the neck.

On admission the nutrition was good, the pulse of fair force, the heart and lungs gave no physical signs, the neck was rigid and drawn backward, the knees were drawn up and resisted extension, there was some muscular twitching, no eye symptoms, alternating restlessness and unconsciousness. The urine contained a little albumin. From the seventh to the eleventh day of his disease there was considerable improvement. His mind became clear, the restlessness and stupor disappeared, and he had some appetite.

From the twelfth to the eighteenth day he complained a good deal of headache, but was rational and asked for food.

On the nineteenth day the white blood cells were 14,400.

On the twentieth day there was vomiting and headache, and after this there was steadily progressing emaciation. He lay quietly in bed, sometimes restless, but gradually became more stupid until he died on the fiftieth day of his illness.

(For chart of Case XXI, see Fig. 10.)

Autopsy.—The cortex of the brain is intensely congested. There is a purulent exudate in the pia mater along the course of the vessels, with small pockets of pus in the pia over the convexity. The lateral ventricles are distended with serum. The other viscera are normal, except for some congestion of the lungs and liver.

Case XXII.—Female, 16 years old, May 16, 1900. The girl arrived in this country from Sweden twelve days ago. Until the present time her health has always been good.

Thirty-six hours before admission she was suddenly attacked with vomiting, chills, sweating, and very severe headache. The next day the vomiting continued and she was alternately unconscious and actively delirious.

Now her nutrition is good, conjunctivæ inflamed, face flushed, no physical signs over the chest and abdomen, rigidity and

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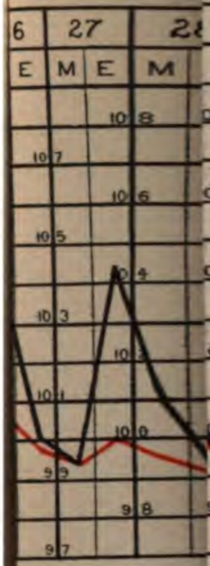

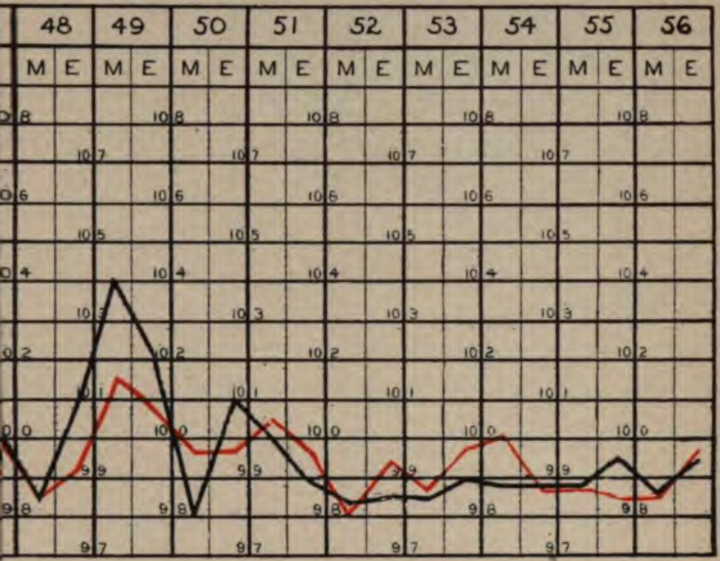

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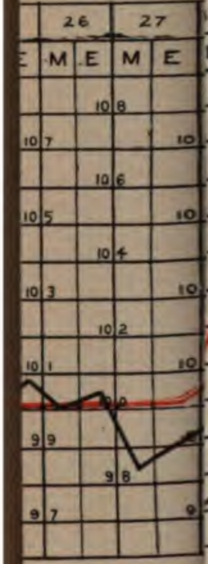

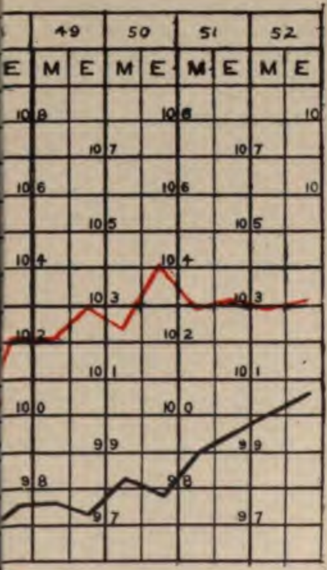

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tenderness of the muscles of the back of the neck, the pupils normal, occasional strabismus, unconscious, groaning as if from severe pain. Urine 1009, no albumin, a few granular casts.

On the fourth day of her illness strabismus and nystagmus were marked, the back of the neck rigid and painful; she continued unconscious and groaning. On the sixth day the unconsciousness and groaning continued, there was marked opisthotonos, a little twitching of the muscles of the face, a fine papular eruption over the upper part of the body.

On the eighth day the delirium and groaning ceased, the stupor was not as profound, she lay in the position of opisthotonos. On the eleventh day she was evidently better; very little stupor, could understand questions, but the opisthotonos continued.

On the thirteenth day cultures from the fluid of a lumbar puncture gave a pure culture of the diplococcus intracellularis. After this there were no more cerebral symptoms and she improved in every way, except that she continued to have an afternoon fever.

On the forty-eighth day she began to have scraped beef to eat, and on the forty-ninth day she was taken out of bed for the first time. After this convalescence was rapid and she was discharged from the hospital in good condition on the sixty-fourth day.

Apparently the meningitis ran its course by the twenty-fifth day, and after that she had a septic temperature until the forty-ninth day, when she was taken out of bed.

(For chart of Case XXII, see Fig. 11.)

2. *The Obscure Cases.*—The symptoms come on gradually; some of the patients are not in bed until after several days, none of them look very sick for the first one or two weeks.

In some of the patients the first symptom has been a headache like that of malaria or of syphilis.

In others, besides the headache, there have also been loss of appetite, vomiting, photophobia, drowsiness, sleeplessness, tenderness over the back of the neck, or an eruption like that of typhoid.

In still others there has been no headache, only prostration, sleeplessness, and a little fever.

For one or two weeks, or even longer, the patients do not look very sick. They make you think of malaria, or syphilis, or a mild typhoid. But they do not get any better, they lose flesh and strength, develop alternating stupor and delirium, and finally die after from four to eight weeks.

The temperature is seldom over 102° F., often down to 99° or even 97° F.

The pulse is between 70 and 120. After death, in most of the patients, the lesions found have been those of cellular meningitis, but in one case there was a large exudation of fibrin and pus at the base of the brain. These cases have interested me very much on account of their long duration and the indefiniteness of their symptoms.

Case XXIII.—Male, 42 years old, March 14, 1881. The man had a chancre and buboes fifteen years ago, but has had no secondary symptoms. He has been intemperate. For several years he has had occasional cardiac palpitation, dyspnœa, and œdema of the feet. One week ago he began to have fever, headache, loss of appetite, pains in the bones, and prostration.

Now he looks fairly nourished, somewhat prostrated, tongue clean, pupils normal, conjunctivæ congested, his mind is dull and stupid.

The temperature ranged from 101°-103° F., the pulse from 92-128, the breathing from 30 to 36. He continued to become weaker and more stupid, but without any characteristic symptoms of any particular disease, until the thirteenth day of the illness, when he died.

Autopsy.—There is an old thickening of the pia mater along the course of the blood vessels. The whole pia looks sodden, but is not congested, nor is any fibrin or pus visible. It is, however, everywhere infiltrated with large numbers of polygonal cells and leucocytes.

There is an old thickening of the mitral valves of the heart. The lungs are emphysematous. The stomach shows the changes of chronic gastritis. The kidneys weigh together nine ounces. The epithelium of the convoluted tubes is swollen.

It was evident that this man was suffering from an infectious disease, but a positive diagnosis was not possible without the autopsy.

Case XXIV.—Female, 11 years old, January 11, 1888. Her mother, an uncle, and an aunt died of phthisis.

Five days ago she began to have severe pain in the left frontal region, lost her appetite, and the next day began to vomit everything she ate. She was dull and drowsy in the daytime and restless and sleepless at night.

Now she is fairly nourished, her face flushed, conjunctivæ congested, tongue moist and nearly clean. Urine 1014, no albumin or casts. There are no physical signs over the heart or lungs.

The patient continued to have headache, vomiting, and constipation; sometimes better, sometimes worse. She gradually lost flesh and strength, and died on the forty-second day of her illness.

(For chart of Case XXIV, see Fig. 12.)

Autopsy.—The pia was congested and lustreless. No serum, fibrin, or pus was evident to the naked eye, but the minute examination showed a well-marked infiltration with polygonal cells and leucocytes.

In the lungs the large and small bronchi contained pus. The rest of the viscera were normal.

This case while it was going on gave me the impression of an obscure tubercular meningitis.

Case XXV.—Female, 28 years old, March 2, 1889. Three weeks ago she began to complain of severe headache. For a week she continued to keep about; since then she has complained of severe pain in the head and epigastrium and of constipation.

Now fairly nourished, face dusky, tongue dry, in a condition of stupor with muscular twitchings. Urine 1016, a trace of albumin. She looks like a person with uræmic or alcoholic poisoning. There are subcrepitant râles over both lungs.

The patient remained in the same condition of alternating stupor and delirium, with muscular twitchings, lying on her back, steadily emaciating, until she died on March 12, the thirty-first day of her disease.

(For chart of Case XXV, see Fig. 13.)

Autopsy.—There is still a thick layer of subcutaneous fat. The pia mater over the convexity is thick and finely roughened and infiltrated with polygonal cells and leucocytes. The pia over the base is also infiltrated with fibrin. The lungs are œdematous. The other viscera are normal.

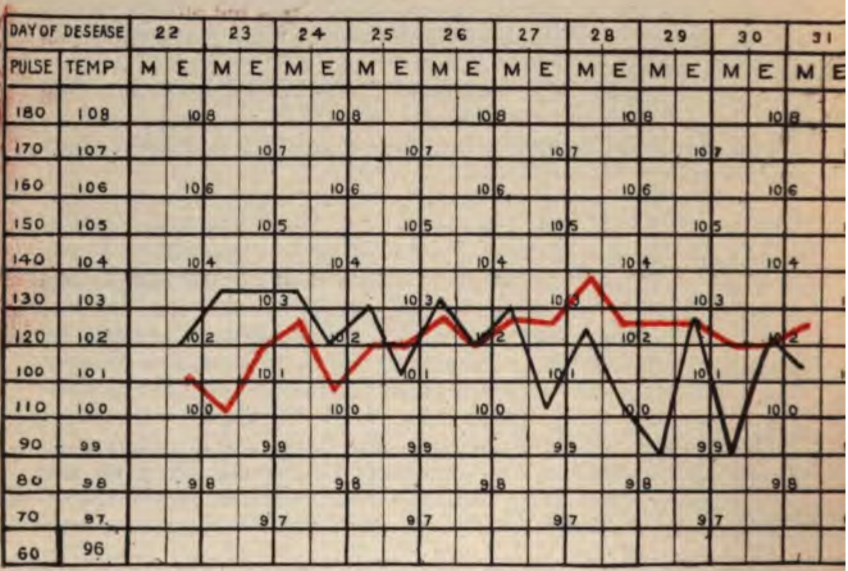
Case XXVI.—Male, 13 years old, November 4, 1884. Three weeks ago he had a severe chill, followed by fever and sweating, with frequent vomiting and diarrhœa. The chills and fever were repeated every day for two weeks, then they stopped and he felt well until November 5, when he was seized with severe abdominal pain and vomiting.

Twenty-second day: He is still vomiting; the bowels have not moved for three days; he complains of severe pain in the abdomen, but does not look bad.

Twenty-third day: He had repeated convulsions, violent delirium, crying out loudly, with intervals of profound stupor. There was tenderness and contraction of the muscles of the back of the neck.

Twenty-fifth day: The active cerebral symptoms continue, there is no more vomiting, the urine and fæces are passed involuntarily.

FIGURE 13.



Temperature black; pulse red.

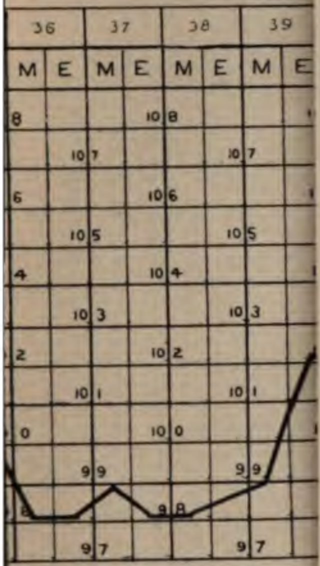
Twenty-eighth day: Rather better, no more active delirium, but profound stupor.

Thirty-third day: The same condition of profound stupor, with intervals of restlessness and crying out, but he also has short intervals of intelligence.

Thirty-fifth day: His condition is the same, but he takes his food and medicines better.

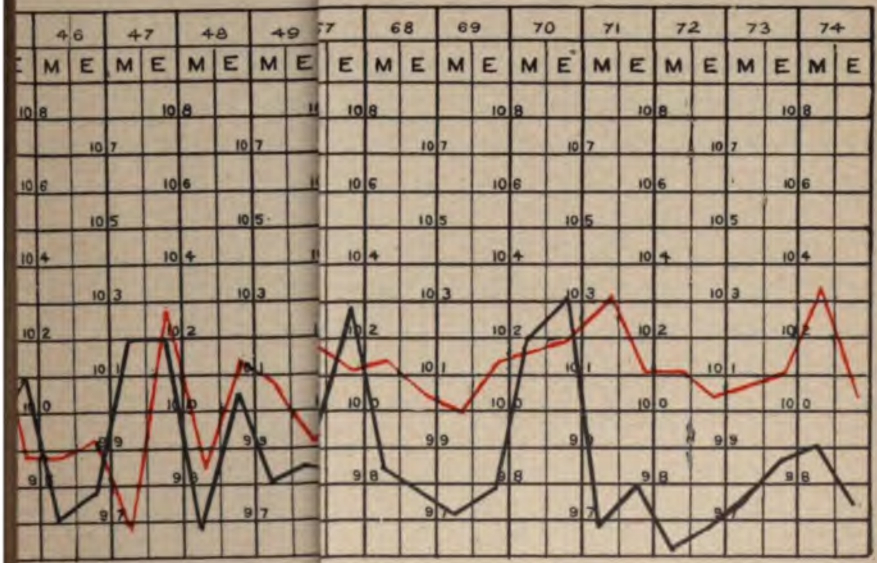
Fortieth day: He is still stupid most of the time, but can eat solid food.

Figure 14.



Temperature Chart.

Figure 15.



rature black; pulse red.

Forty-third day: He is not as well and again has active delirium, but still eats solid food.

Forty-eighth day: He is better, with much longer rational intervals and a ravenous appetite.

Eightieth day: The temperature has been normal for some time, he has gained flesh and strength; his rational periods are more frequent and longer, and to-day, for the first time, he is out of bed.

After this he continued to improve until he was entirely well. (For chart of Case XXVI, see Fig. 14.)

Case XXVII.—Female, 11 years of age, August 7, 1882. Three weeks ago she had fever, pain in the head and ears, then pain in the left iliac region, and soon afterward vomited four round worms.

After this she had pains in the head, ears, and side, with an irregular fever and sometimes delirium.

Thirtieth day: Since her admission she has continued to complain of pain in the ears; the pupils are dilated; she is restless, moaning and crying out with pain; the tongue is dry and coated, there is vomiting and general hyperæsthesia with tenderness and stiffness of the muscles of the back of the neck; all the joints are flexed and somewhat rigid. There is nothing the matter with the ears, and the pain she refers to them seems to be really a headache.

Forty-fourth day: The headache, hyperæsthesia, delirium, crying and moaning, and occasionally vomiting have continued.

Sixty-ninth day: Since the forty-fourth day there have been intervals of quiet between the periods of pain and delirium. These intervals have become longer and longer, until by the seventy-sixth day she is apparently nearly well. She was left very weak and emaciated, and it was not until the one hundred and twentieth day that she was well enough to leave the hospital. (For chart of Case XXVII, see Fig. 15.)

Chronic Meningitis.

True chronic meningitis is a very real and serious lesion. It is not to be confounded with the simple opacities and thickenings of the pia mater, which are so common in adult life.

Lesions.—Either the pia mater of the base of the brain alone may be inflamed (basilar meningitis), or the pia mater over the convexity alone, or the entire pia mater, or circumscribed patches of the membrane. The pia mater is thickened and opaque, the thickening sometimes being very considerable. There is a formation of new connective tissue and a production of pus, fibrin, and serum; the relative quantity of these inflammatory products varies in different cases. Firm and extensive adhesions may be formed between the dura mater and the pia mater. Not infrequently the cortical portions of the brain participate in the morbid process, and we find an infiltration of small spheroidal cells around the blood vessels, thickening of the walls of the vessels, and degenerative changes and atrophy of the brain tissue. New connective tissue may also form in the brain substance, and the latter may become adherent to the pia mater. The ventricles of the brain may be much dilated and distended with serum, their ependyma may be thickened and roughened.

Causes.—Chronic meningitis may be caused by blows on the head and by fractures and inflammations of the cranial bones. Sometimes the injury to the head antedates by several years the symptoms of the meningitis. It is often associated with pachymeningitis, with tumors of the brain, and with chronic arteritis of the cerebral arteries. It is the direct result of syphilis. It occurs with chronic nephritis, with chronic alcoholism, and in persons who are badly nourished and of dissipated habits. It is regularly found in the general paralysis of the insane.

Symptoms.—The course of the disease is that of an inflammation, chronic from the outset, and with exacerbations from time to time. The symptoms are at first obscure and intermittent, and vary much in different individuals as to their number and distinctiveness. So the diagnosis is always difficult and sometimes quite impossible.

The patients complain of more or less headache—continuous or intermittent. The mental faculties and the temper gradually deteriorate, and such a change may go on to complete insanity. From time to time there are attacks of unconsciousness, of stupor, of delirium, of muscular contractions or rigidity, of aphasia, of general convulsions, or of vomiting. After a time the nutrition is impaired, and there is a gradual loss of flesh and strength. At the time when the symptoms are most marked there may be a moderate rise of temperature.

Chronic meningitis usually lasts for a number of years, but it may happen that the early symptoms are slight and that at some time severe symptoms are suddenly developed, as if of an acute cerebral lesion.

The patients die from some intercurrent disease, or become insane, or die with marked cerebral symptoms or in a condition of emaciation and exhaustion.

Treatment.—The first point to determine is whether the patient has had syphilis. If this is the case, the use of mercury and the iodide of potash may be of much service. In the cases due to other causes there seems to be no direct treatment for the meningitis. The improvement of the general health and the alleviation of symptoms must be attempted, but, as a rule, the patients get worse.

Case XXVIII.—Female, 45 years old, January 24, 1869. This woman was in the hospital under observation for nearly a month. She was suffering from chronic rheumatism; she was rather dull and sleepy, but could answer questions intelligently. It was evident that she had chronic endocarditis and chronic phthisis, but there were no marked cerebral symptoms.

Autopsy.—The pia mater over the entire surface of the brain is thickened, greenish colored, and succulent. In some places thick pus exudes from the cut membrane. The lateral ventricles contain serum and yellow flakes.

There are vegetations on the pulmonary and tricuspid valves of the heart.

The upper lobe of the right lung is changed into fibrous tissue with old cavities.

Case XXIX.—Male, 40 years old, November 26, 1870. The man has not been well for two years. He first noticed feelings of numbness, beginning in the right thumb and extending over the right side of the body. Two months later he had an attack which caused him to fall to the ground, but without loss of consciousness. He had after this a number of attacks of numbness in the right side of the body, falling to the ground and sometimes losing consciousness. The last attack was six days before his admission to the hospital. During these two years he has not been able to work because his mental faculties were feeble and he was unnaturally drowsy.

He was in the hospital for ten weeks. During that time his condition was pretty good, part of the time childish and drowsy, part of the time fairly rational; not confined to bed. Two days before death he had two convulsions, the following day one convulsion, and the next day he died.

Autopsy.—The pia mater over the entire brain was much thickened, opaque, and infiltrated with serum. There were large vegetations on the aorta two inches above the valves. The lungs were emphysematous. The kidneys showed the lesions of chronic nephritis without exudation.

CASE XXX.—Male, 33 years old, January 8, 1879. Five years ago he had sores on his penis, followed by an eruption and pains in the bones. The pains in the bones have troubled him from time to time since. Three years ago the left eye became blind and seven months ago he began to lose the sight of the right eye. By February 14 both optic nerves had become atrophied.

On August 22 he was suddenly seized with a convulsion, followed by coma, the pulse and respiration were irregular and feeble, the temperature remained normal. During the rest of the day he had sixteen more convulsions, the temperature rose to 104° F., he became comatose and died the next morning.

Autopsy.—The body was fat and well nourished. The pia mater was much thickened by a growth of new connective tissue. There were no changes in the brain tissue or cerebral arteries. The intracranial portions of both optic nerves were atrophied. The other viscera were normal.

CASE XXXI.—Male, 27 years old, December 20, 1889. Intemperate, had a chancre eight years ago, has had occasional morning vomiting. Three years ago he fell and hurt the back of his head.

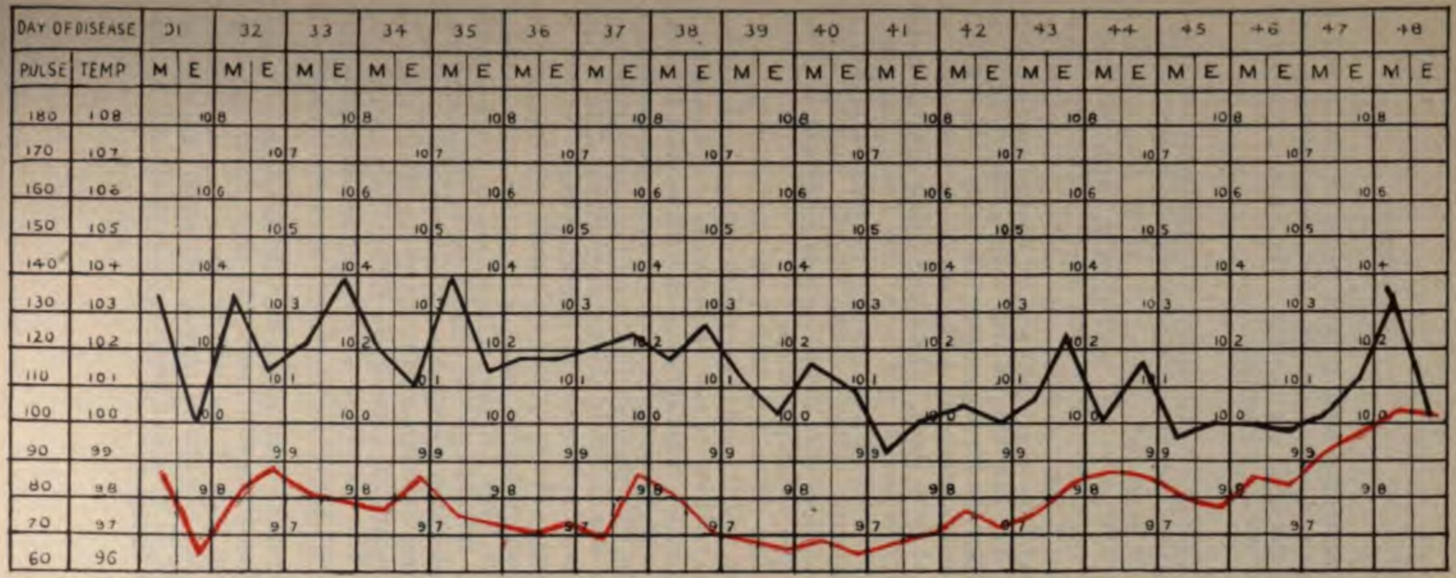
One month ago he began to have severe pain in the head and back of the neck, worse at night. There was no vomiting, no chills; he was constipated and sleepless. Now he is fairly nourished, of good color, his tongue moist and coated, his appetite good; complains bitterly of the headache and inability to sleep. Urine 1010, a trace of albumin.

Fortieth day: The headache has continued in spite of a variety of drugs. The man is getting worse and actively delirious. The delirium continued until he died on the fiftieth day.

(For chart of Case XXXI, see Fig. 16.)

Autopsy.—The surface of the brain is flattened. The pia mater over the convexity is congested and lustreless. The pia over the base of the brain and over the posterior surface of the

FIGURE 16.



Temperature black; pulse red.

cord is infiltrated with a thick layer of fibrin and pus. The lateral ventricles are dilated and distended with serum. The bones of the skull are normal. The viscera are normal, except for some venous congestion.

Case XXXII.—Complicated with inflammation of the cerebral arteries and dilatation of the lateral ventricles.

Male, 69 years old, October 27, 1882. The patient was a silk manufacturer, who had worked very hard, but had been well until a few weeks before October 27. Then he began to have symptoms of cystitis, with considerable pain about the bladder. These symptoms continued and he became restless and sleepless. When I first saw him he was complaining a good deal of the cystitis, but the striking thing about him was the sleeplessness and extreme irritability. He was fairly nourished, intelligent, gave no physical signs, had some gastric dyspepsia and flatulence, was not confined to bed or to the house, but had a feeble, shuffling, uncertain gait; unable to sleep at night; during the day constantly talking about himself, anxious, unable to sit still. I thought that he had chronic inflammation of the cerebral arteries and would get worse.

November 30 he left New York and went home in the same condition, and there was very little change until February, 1885. Then the restlessness and sleeplessness were succeeded by mental imbecility and sluggishness. His urine was frequently examined; it was of normal specific gravity and contained no albumin. He continued in this condition until February 1, 1886. Then he was suddenly seized with convulsions, which were repeated until he died on the following day.

Autopsy.—The body was well nourished and there was a thick layer of subcutaneous fat.

All the large arteries at the base of the brain showed the irregular thickenings and dilatations of chronic arteritis.

The pia mater throughout was opaque and thickened; its inner layers were infiltrated with numerous small, round cells. The lateral ventricles were much dilated and distended with clear serum. The ependyma of the ventricles showed no changes; the septum was thinned. There were no changes in the brain tissue.

The bronchi were congested and there were irregular areas of red hepatization. The aortic and mitral valves were thickened; the wall of the left ventricle was thickened; the aorta showed the changes of chronic arteritis; the kidneys were far advanced in chronic nephritis, with thickened arteries (arterio-sclerotic kidney).

THE VENTRICLES OF THE BRAIN.

Inflammations of the pia mater are often accompanied by lesions of the ventricles of the brain. Less frequently we find lesions of the ventricles occurring by themselves with little or no change in the pia mater. We distinguish:

Acute ependymitis.
Acute dilatation of the ventricles in adults.
The chronic hydrocephalus of children.

1. ACUTE EPENDYMITIS.

Of this we find two varieties—an acute and a subacute form.

In the acute form we find the ependyma congested, infiltrated with pus cells, and coated with a layer of fibrin and pus. The ventricles contain purulent serum.

The patients exhibit a decided febrile movement, general convulsions, alternating stupor and delirium, local paralyses, and finally coma. The disease runs its course within a few days. The symptoms resemble those of an acute tubercular meningitis. So few of these cases have been observed that we are ignorant of the prognosis. The treatment would seem to be the same as that for acute meningitis.

In the subacute form the ventricles are dilated and full of serum; the ependyma is thickened; the endothelial cells on its surface are multiplied, and there is a growth of cells around the blood vessels.

The patients have headache, vomiting; at first photophobia, later dilated pupils; hyperæsthesia of the skin, an irregular rise of temperature, finally coma.

The disease lasts for weeks or months; the symptoms resemble those of a subacute tubercular meningitis.

These cases again are so seldom recognized that we do not know how many of them may recover.

2. Dilatation of the Ventricles of the Brain. Serous Apoplexy. Meningitis Serosa. Hydrocephalus.

This condition of the ventricles—a condition which gives marked symptoms and causes death—is obscure to the last degree. It occurs, as I have seen it, both in strong and vigorous adults and in persons who have suffered from chronic diseases. In some persons there is a previous history of injuries to the head, or of mental or bodily overwork. It occurs both in laborers and in professional and business men. In New York the cases are not infrequent.

Many cases are scattered through medical literature. Abercrombie, in his work on the brain in 1843, gives a very good description of the condition, with excellent illustrative cases. He calls it serous apoplexy.

Quincke, in a monograph published in 1893, describes the condition under the name of meningitis serosa. He distinguishes cases with an acute invasion followed by an acute or a chronic course, and chronic cases running a chronic course with or without exacerbations.

Of the acute cases he gives six examples, all terminating in recovery; three of these were in young women and two in young children. None of them seem to me to be certain examples of this disease.

Of the acute cases with chronic course he gives one case with autopsy, which is apparently tubercular.

Of the chronic cases there are only two which are probably examples of this condition; the others seem to be cases of tubercular ependymitis or of ordinary chronic hydrocephalus.

Lesions.—Most of the cases I have seen have been fatal ones with autopsies. Without an autopsy I do not feel certain of the diagnosis. Cases in which chronic meningitis or miliary tubercles in the ependyma exist belong to a different category, as do the cases of moderate serous effusion with chronic diseases.

In the characteristic cases the surface of the brain is flattened; there are no marked changes in the pia; all the ventricles are dilated and distended with clear serum; the septum lucidum is thinned; the ependyma is white, somewhat thickened, its surface smooth or finely granular, or it is entirely normal. There are no evidences of inflammation in the ependyma. There is no anatomical change present that at all accounts for the production of clear serum in such quantities as to largely distend the ventricles. Less frequently the distension is confined to the lateral ventricles, and occasionally to one lateral ventricle.

Symptoms.—The patients suffer from headache, noises in the ears, loss of memory, dulness of the mental faculties, slowness of speech, sleeplessness, disturbed digestion, sometimes temporary aphasia or local paralyses. These symptoms may only last for a few hours, or they may continue for a number of weeks. Then, either rapidly or gradually, they pass into the condition of complete coma. In this condition they remain for a few days and then die, the temperature rising before death.

Others, on the contrary, develop an active delirium, blindness, internal strabismus, convulsive movements of groups of muscles, a febrile movement, and finally become comatose.

Case I.—Male, 40 years old, February 13, 1885. The patient has always been a healthy, hard-working man and has received no injury.

Five weeks ago he began to have a severe frontal headache, loss of memory, deafness, apathy, and was unwilling to move. He has remained in this condition up to the present time. Now the temperature, pulse, and respiration are normal, the pupils are unequally dilated, the hearing is dull; he swallows easily; no paralysis, no disturbances of sensation; the tendon reflexes are exaggerated. The man lies quietly in bed in an apathetic condition. It was thought that he was suffering from pressure on the brain, probably due to a tumor.

He remained in the condition of stupor alternating with mild delirium, and then became comatose. On February 16 the temperature suddenly rose to 103° F., and consolidation of the lower lobes of both lungs was made out. He died on February 22.

Autopsy.—The dura mater and pia mater and the cranial bones are normal. The cerebral arteries and veins are normal. Both lateral ventricles are widely dilated and distended with clear serum. There are no changes in the ependyma of the ventricles. The heart and large vessels are normal. In both lungs there is general purulent bronchitis and broncho-pneumonia of the lower lobes. The kidneys showed the lesions of the early stages of chronic nephritis.

CASE II.—Male, 26 years old, April 5, 1880. The man has always been strong and healthy. Eleven years ago he fell about thirty feet and struck on his head and left side. He was carried home insensible; the next morning he was conscious, but had no power in his legs. He gradually got better, and after two weeks went back to work. He has been accustomed to carry heavy weights of slate on his head and often has headaches in the evening. Seven weeks ago he began to have lightness and pain in the head during the daytime.

Five weeks ago the pain became more frequent, compelling him to leave his work and go home. The pain now came on in attacks, with dizziness, severe pain in both cheeks, nose, and jaws, about four attacks a day. Later there was pain in the back of the head and down the spine.

April 4, in the night, he had a severe attack of pain in the head and noises in the ears.

From April 5 to 9 he had six or seven attacks of pain a day. During these attacks he would hold his breath, make faces, look idiotic, and have slight internal strabismus. He died suddenly in the night of April 9.

Autopsy.—The bones of the skull contain an unusual amount of dark blood. The surface of the brain is flattened. Minute examination of the pia shows no changes in it. All the ventricles of the brain are much dilated and filled with clear serum. The septum between the lateral ventricles is thin and transparent. The ependyma of the ventricles is normal. The brain is anæmic; the cerebral arteries are normal.

All the viscera were examined and showed no changes except those of venous congestion.

3. THE CHRONIC HYDROCEPHALUS OF CHILDREN.

Lesions.—There is an accumulation of serum in the ventricles of the brain so large in quantity that the brain is thinned, the dura mater stretched, and the cranial bones separated from each other. The ependyma appears to be normal or is somewhat thickened.

Causes.—We are ignorant of the causes of this lesion. It seems to begin during intrauterine life; it may reach a considerable development before birth, or remain latent for months or years after birth.

Symptoms.—If the disease is so much developed during intrauterine life that the large head prevents the birth of the child, craniotomy must necessarily be performed.

If the disease is moderately developed during intrauterine life the child can be born alive, but it is at once evident that the head is too large. The child does not perform its functions and dies in a short time.

In many cases, at the time of birth, the child shows no evidences of the disease, and it is not till after months or years that its symptoms are developed. Of these later developed cases we may distinguish a mild and a severe form of the disease.

In the mild cases the head is usually large, the fontanelles and sutures open, the face small. The intelligence remains good. From time to time, for periods of several days, the child suffers from disturbances of digestion and nutrition, a febrile movement, fits of crying, restlessness, and drowsiness.

In these mild cases the disease is not of itself fatal, but the children are apt to be carried off by some of the diseases of childhood.

In the severe cases there is marked impairment of nutrition; the children are dull and stupid, irritable and drowsy. There are often strabismus, nystagmus, dilated pupils, protruded eyeballs, blindness, deafness, general convulsions, and paraplegia. There may be a febrile movement. These symptoms are interrupted by periods of improvement, but the course of the disease is usually downward, and the children die in an attack of convulsions, or of inanition, or of some intercurrent disease. Rarely they grow up to adult life, but remain more or less idiotic.

The treatment is mainly directed to the general condition of the patient. Careful feeding, a clean skin, good air, the administration of iron and of cod-liver oil, are essentials. During the exacerbations of the symptoms the iodide of potash may be of service. A variety of surgical procedures have been tried, but none of them are satisfactory.

INFLAMMATION OF THE CEREBRAL ARTERIES.

The arteries throughout the body are frequently the seat of chronic inflammation. This condition is considered by many to be a degeneration and is described under the name of atheroma, fibrosis, or sclerosis. To me, however, it seems a true chronic productive inflammation, caused by different irritant poisons. It occurs most frequently in the aortic system of vessels, and may involve the aorta alone, or the aorta with many of its branches, or the arteries of some particular part of the body.

Of the cerebral arteries the larger vessels at the base of the brain are the ones most frequently involved, but the lesion may be so extensive that hardly any part of the cerebral arterial system escapes changes.

The changes are as follows:

1. There is an increase in the size and number of the endothelial cells. This is the only change. It does not lead to obliteration of the artery. This is best seen in the small arteries surrounded by miliary tubercles or by small gummata.

2. There is a growth of new connective tissue, apparently originating in the endothelium, which narrows the lumen of the artery and may finally occlude it. The growth is composed of branching cells, small round cells, and basement substance; later the cells become smaller and less numerous, the basement substance denser. The new tissue grows inward, so as to fill up more and more the lumen of the artery, until finally the vessel may be almost entirely occluded. This is often called obliterating endarteritis.

3. There is thickening of the inner coat beneath the endothelium. The change begins by a growth of cells and a splitting up of the basement substance in the intima immediately beneath

the endothelium. As this growth continues there are fewer cells and more basement substance. In this way the inner coat is thickened and the lumen of the artery narrowed. These changes may occur by themselves or there may be, in addition, thickening of the muscular and outer coats, replacement of the muscular coat by dense connective tissue, thinning of portions of the arterial wall and irregular dilatations, deposition of the salts of lime in the new tissue.

As a result of these changes the arteries are rendered very irregular channels for the passage of the blood—narrowed in some places, dilated in others, or completely occluded. The blood supply of the corresponding portions of the brain is variable or cut off altogether.

The diseased arteries may rupture, with extravasations of blood. Portions of the brain may become softened from the occlusion or thrombosis of the arteries. Either small or large aneurisms may be formed.

Chronic arteritis follows the laws which govern all the chronic productive inflammations: It belongs to people over forty years old; it is caused by the poisons of syphilis, of gout, and of other unknown toxins; it is frequently associated with one or more of the chronic productive inflammations of other parts of the body—emphysema, chronic endocarditis, and chronic nephritis; the patients are liable to attacks of contraction of the arteries; the anatomical changes can exist for years without clinical symptoms.

There is a good deal of variety in the behavior of patients with cerebral arteritis. We distinguish:

1. The cases in which we find diseased cerebral arteries after death without any history of cerebral symptoms during the life of the patient.

2. The cases with a definite syphilitic history, which at some time begin to have severe and persistent headaches, accompanied with disturbances of speech or paralyses of groups of muscles. These patients recover either completely or partially with antisyphilitic treatment, and after recovery it is often difficult to tell whether the symptoms have been caused by gummata or arteritis.

3. Cases with a syphilitic history, which give marked cerebral symptoms continuing up to death, and after death either cerebral arteritis alone or arteritis with areas of softening of brain tissue is found.

In these patients headache is regularly the first symptom, then come the aphasia, the paralyses of groups of muscles, the attacks of unconsciousness and delirium, and finally death, all within a few weeks.

CASE I.—Male, 40 years old, October 21, 1884. Eight months ago the man had a chancre on the penis which lasted four months. Three months later a roseola came out and lasted for nine weeks. For the past nine weeks there have been mucous patches in the mouth.

Seven weeks ago he began to have sleeplessness and headache, which have continued up to the present time. Eight days ago, when he woke in the morning, he found he could not use the right arm and leg.

Now the man is fairly nourished, the skin is covered with a syphilitic eruption, the scar of a chancre can be seen on the penis, there are mucous patches on the lips and mouth. The left arm and leg are partially paralyzed without loss of sensation.

October 23: The man fell out of bed and became unable to speak.

October 25: He is becoming stupid and has right hemiplegia.

October 26: He is comatose, with a temperature of 103° F.; before this the temperature was not above 98.6° F.

On October 27, after eight weeks of head symptoms, he died.

Autopsy.—The pia mater is dry and congested. The basilar artery for its entire length shows a marked thickening of its wall and narrowing of its calibre; the right posterior cerebral artery is in the same condition; there are no changes in the brain.

In the lungs the bronchi contain muco-pus; the lower lobes are congested, with irregular areas of red hepatization. The rest of the viscera and the arteries show no morbid changes.

CASE II.—Female, 28 years old, December 11, 1879. In November, 1877, she contracted syphilis; in January, 1878, she was treated for secondary symptoms. After this she was well until two months ago, when she began to have headache and pains in the left hip. Six weeks ago she suddenly became aphasic and the left side of the face became paralyzed. Soon after this she became unconscious for three days and then was mildly delirious for a week. Three days ago her right side became paralyzed. Her eyesight has remained good. She has been unable to pass her urine since the first attack of paralysis.

Now the woman is well nourished, her appetite is good, her tongue is clean, her bowels are regular, the pulse is 90, respiration 28, temperature 100.2° F. The urine is 1020 and contains a trace of albumin. The right side of the face is paralyzed; in the right arm there is complete loss of motion with impaired sensation; in the left arm sensation is impaired; in the right leg motion is lost, sensation is impaired; in the left leg sensation and motion are good.

December 16: She can move the right leg and arm a little; there is less paralysis of the right side of the face, but there is some difficulty in swallowing.

December 22: In the tenth week of her cerebral symptoms she became comatose, the temperature rose to 105° F., and she died.

Autopsy.—The bones of the skull and the dura mater are normal. The surface of the pia mater is dry and lustreless, its blood vessels are congested. The basilar and middle cerebral arteries show the marked changes of chronic arteritis. There is softening of the posterior half of the ventricular surface of the left corpus striatum, of the entire left lenticular nucleus, and of the internal capsule between the corpus striatum and optic thalamus on the left side. None of the other viscera show morbid changes of any consequence.

4. Cases without a syphilitic history, usually of considerable duration, the symptoms coming on in attacks, the patients dying in such an attack or from some intercurrent disease.

These cases are often seen and are of much clinical importance. The symptoms are at first in the form of temporary attacks, which last for hours or days and then disappear. As a rule each attack is accompanied by contraction of the arteries.

Such attacks are repeated at irregular intervals during months or years, the patient presenting few or no cerebral symptoms during the intervals. As time goes on the attacks become more severe and more frequent. It is, however, possible for the patient to die during the first attack.

The temporary attacks are not all of the same character.

There may be only headache or anxiety, restlessness and insomnia, or vertigo, or aphasia, or hemianopsia, or loss of consciousness, or general convulsions, or spasmodic contractions of groups of muscles, or paralysis of one arm, of one leg, or of the whole of one side of the body, or loss of sensation in different areas of the skin.

The first attack may prove fatal, but the patient often continues to live for months or years. Even after recovering from the first attack, however, there is a perceptible change in the patient's mental and bodily condition, sometimes very marked. The attacks are repeated, until finally the patient develops the symptoms of chronic meningitis, or becomes completely hemiplegic, or dies with general convulsions or coma.

CASE III.—Male, 38 years old, December 29, 1882. Eighteen months ago he had an attack of paralysis, and since then has at times been troubled in his speech. He recovered from the paralysis and was able to go back to his work.

December 29, while at work, he complained of headache and dizziness, and then gradually lost the power of motion and speech and became completely comatose with stertorous breathing; a full, hard pulse; temperature 101° F.; urine 1026, a considerable quantity of albumin. He remained completely comatose, the temperature rising till it reached 109° F., and died two days later.

Autopsy.—The dura mater and pia mater are normal; the brain tissue is normal; the basilar artery is nearly occluded by the patches of thickening of a chronic arteritis.

In the lungs the bronchi are congested and coated with mucus; the lower lobes are congested, with irregular areas of red hepatization.

The kidneys show the early stages of the atrophic form of chronic nephritis.

CASE IV.—Female, 69 years old, April 14, 1888. The patient had a similar attack about a year ago, from which she recovered. One week ago she developed complete loss of motion on the left side. She was neglected and starved, so that when she was brought into the hospital her general condition was very bad.

There was complete loss of motion on the left side, but good sensation. She was feeble and emaciated; could answer questions; temperature 101° F.; urine 1024, a trace of albumin. She remained hemiplegic, but did not become comatose, and died ten days later.

Autopsy.—The dura mater, the pia mater, and the brain are normal. The basilar artery, both middle cerebral arteries, and both posterior cerebral arteries have their walls irregularly thickened and thinned, with narrowing and dilatation of their lumen.

In the lungs there is fibrin on the pleura of both lower lobes, there is irregular consolidation of the upper and lower lobes of the right lung. In the heart the aortic valve is thickened with vegetations; the mitral valve is thickened and stenosed; the left ventricle is hypertrophied. The whole length of the aorta shows the advanced changes of chronic arteritis.

The kidneys are atrophied.

5. Cases of the same character as those just described, but the fatal attack is accompanied with softening of portions of the brain tissue.

These cases cannot be distinguished clinically from the cases of cerebral arteritis without softening. In speaking of softening of the brain it is to be understood that I made the autopsies myself a few hours after death.

Case V.—Male, 50 years old, September 24, 1882. The man is a sailor. Ten days ago, while sitting on deck, he became dizzy and fell down, but did not lose consciousness. After this he felt well again and could go about and up the rigging.

One week ago his walk became affected. He would go forward or backward faster than he wished. He had several attacks of vertigo, could not go upstairs without falling, and complained of frontal headache.

Four days ago the left foot began to drag, and as he walked he would run into people.

Now he is perfectly conscious and walks with a little assistance, although the left leg drags. He speaks with some difficulty; there are no physical signs of disease of the heart or lungs; there is a little paralysis of the left side of the face and partial paralysis of the left leg, without loss of sensation; urine 1025, no albumin or casts.

Thirteenth day: The left leg is weaker, the left arm is getting weak; he talks with more difficulty, complains more of headache, and is unable to get out of bed.

Eighteenth day: The left side is completely paralyzed and he is nearly comatose.

Twenty-second day: There is complete coma, with twitchings of the paralyzed muscles.

Autopsy (ten hours after death).—The pia mater was somewhat congested. All the arteries at the base and their branches present numerous points where the wall of the artery is thickened and its lumen narrowed. In the right middle cerebral artery there is almost complete occlusion. In the right hemisphere the white matter external to the corpus striatum and optic thalamus is soft and of reddish-yellow color. The other viscera show no lesions.

Case VI.—Male, 30 years old, November 12, 1883. Yesterday he began to have severe headache, felt dull and stupid, and in the evening lost consciousness.

To-day the man is semi-comatose, with complete left facial paralysis and hemiplegia. The paralysis of motion is complete, sensation is unimpaired, the reflexes are increased. Temperature 98.6° F., pulse 70. In the evening he was brighter and could answer questions.

Fourth to the thirteenth day: The man lies quietly in bed, stupid, but can answer questions, and can move the left leg a little.

Twenty-fifth day: The man is sometimes quite intelligent, sometimes stupid; he can move the left arm and leg.

Thirty-sixth day: He is out of bed and can walk a little.

Fortieth day: He is back in bed again, very stupid, and the left hemiplegia has returned.

Fiftieth day: He became comatose and remained so, without rise of temperature, until he died on the fifty-sixth day.

Autopsy.—The large arteries at the base of the brain showed a number of areas of white thickening and narrowing. A small part of the anterior portion of the right caudate nucleus and part of the internal capsule beneath it were softened. The other viscera were normal.

Treatment.—In the cases with a distinct syphilitic history energetic antisyphilitic treatment is indicated, especially the use of large doses of iodide of potash. In the long-continued, non-syphilitic cases with repeated attacks, a great deal can be done with arterial dilators. I have seen patients kept fairly well for years by the use of nitroglycerine, chloral hydrate, or potassium iodide whenever the arteries became contracted and the cerebral symptoms threatening.

THROMBOSIS OF THE CEREBRAL ARTERIES AND VEINS.

This is one of the rare cerebral lesions. The principal symptoms are: headache, delirium, stupor, and paralyses of groups of muscles. The causation of the condition is very obscure. I have seen only a few cases, in all of which I made the autopsies myself.

CASE I. *Thrombosis of the Right Internal Carotid and Middle Cerebral Arteries.*—Male, 46 years old, January 19, 1879. For three months the man has been confined to bed with rheumatism, pains in the knees and ankles. Three days ago while walking across the floor he fell down, did not lose consciousness, but the left arm and leg became paralyzed; no loss of speech. The next day the paralysis was a little better, the mind was wandering.

Now the man is fairly nourished, actively delirious, with almost complete paralysis of the left arm and leg; the temperature normal.

He continued to be delirious; the urine and fæces were passed involuntarily. On February 1 the temperature was 103° to 104.8° F., the pulse 120 to 170, the breathing 36 to 84.

On February 2 the temperature rose to 107.8° F. and he died.

Autopsy.—The arteries at the base of the brain are a little thickened. There is a thrombus in the right internal carotid extending into the middle cerebral artery. The white substance of the right hemisphere beneath the two ascending convolutions and the convolution just behind them is markedly softened.

In the lungs there is a diffuse broncho-pneumonia; the other viscera show no lesions.

CASE II. *Thrombus of the Basilar Artery.*—Male, 45 years old, February 21, 1887. Three hours before his death he suddenly became comatose, with stertorous breathing, contraction of the right pupil, no appreciable paralysis; urine 1020, trace of albumin. He came partly out of the coma and then died.

Autopsy.—The pia mater is congested. The basilar artery for its entire length is completely filled with a large, firm thrombus. The brain tissue is not softened.

The viscera show no lesions, except some old fibrous tissue at the apex of the right lung.

CASE III. *Thrombosis of the Straight Sinus.*—Female, 20 years old, June 2, 1871. On April 30, after exposure to the sun, she suddenly fell over and became unconscious. Now she is comatose, pulse 72 and full, respiration 28, temperature 99° F.; a loud systolic murmur at the base of the heart; the right arm and leg are paralyzed; there is incontinence of urine. She remained in the same condition until June 7, when the temperature rose to 103.7° F. On June 8 she died.

Autopsy.—The straight sinus is filled with a large, firm, white thrombus extending through the smaller veins into the venæ Galeni and into the branches from the optic thalami and corpora striata. The upper portions of both corpora striata, of the left optic thalamus, and of the fornix are in a condition of red softening.

The other viscera are normal.

CASE IV. *Thrombosis of the Lateral and Straight Sinuses.*—Female, 23 years old, March 4, 1884. For two months she has complained of severe headache.

Now she has severe headache and vomiting; she looks anæmic; she lies with the eyes closed, the pupils equal and active, quiet and stupid; the tongue dry and coated; pulse 70, temperature 98° F.; urine normal.

March 6: She is very restless; there are twitchings of the muscles of the arms and legs. In the afternoon and night there are frequent attacks of contraction and rigidity of the muscles of the arms; she is unable to swallow; pulse 80-128, temperature 100.6°-107° F.; urine contains a good deal of albumin, hyaline and granular casts.

March 7 she died.

Autopsy.—There are no marks of injury, no lesions of the scalp, the skin, the cranial bones, or the neck. In both lateral sinuses, in the straight sinus, and in the venæ Galeni are red and white thrombi, which distend the veins. The fornix, the caudate nucleus, and the lenticular nucleus, with the white matter between them of the right corpus striatum, and both optic thalami, are softened and full of small extravasations of blood. These changes are most marked in the optic thalami.

In the lungs there is congestion and œdema of the posterior portions.

The liver is small and friable, the hepatic cells are swollen and granular. The stomach and intestines are normal. The kidneys look normal, except that they are pale.

CASE V. *Thrombosis of the Straight Sinus and the Right Lateral Sinus.*—Male, 48 years old, a publisher, a man of nervous and excitable temper, easily irritated, sometimes hysterical, habitually overworked, but yet considered himself to be well.

February 26: He complained of indigestion and of occasional headaches in the morning.

March 3 and 4: He had severe supraorbital and facial neuralgia of the left side.

March 7: He complained of severe right hemicrania, was anxious and hysterical, but had no fever. He continued to be miserable, with pains in the head and nervousness, but without fever, until March 10, when he developed alternating stupor and delirium, still without fever. The man continued in the same condition of alternating stupor and delirium, without any paralysis, but looking more and more septic, until March 23, when the temperature rose to 100° F.

On March 31 he died comatose.

Autopsy.—In the straight sinus and the right lateral sinus are soft, pinkish thrombi not filling the vessels. On the right side, over the posterior end of the second frontal convolution, one of the large veins of the pia is distended with a firm red thrombus. A part of this convolution is mottled with small extravasations of blood. Over this the pia is infiltrated with pus and serum. At a number of other points over the convexity the pia is infiltrated with pus and serum.

CASE VI. *Thrombosis of One of the Veins Leading into the Longitudinal Sinus, with Pachymeningitis.*—Male, 32 years old, July 6, 1875. The man had led a wandering life on sea and land, is intemperate, and has had syphilis.

On June 15 he came from Maine to New York to buy goods. After being in the city for a few days he became ill with headache and pains in his bones.

July 2: He was well enough to go to Coney Island by the steamboat. After this day he can give no account of himself.

July 6: He was brought into the cells of the hospital.

July 8: He was removed to the ward. He was very dirty, his urine retained, his tongue coated, swollen, and excoriated, the muscles of the back of the neck stiff. He looked as if he had cerebro-spinal meningitis or typhoid fever.

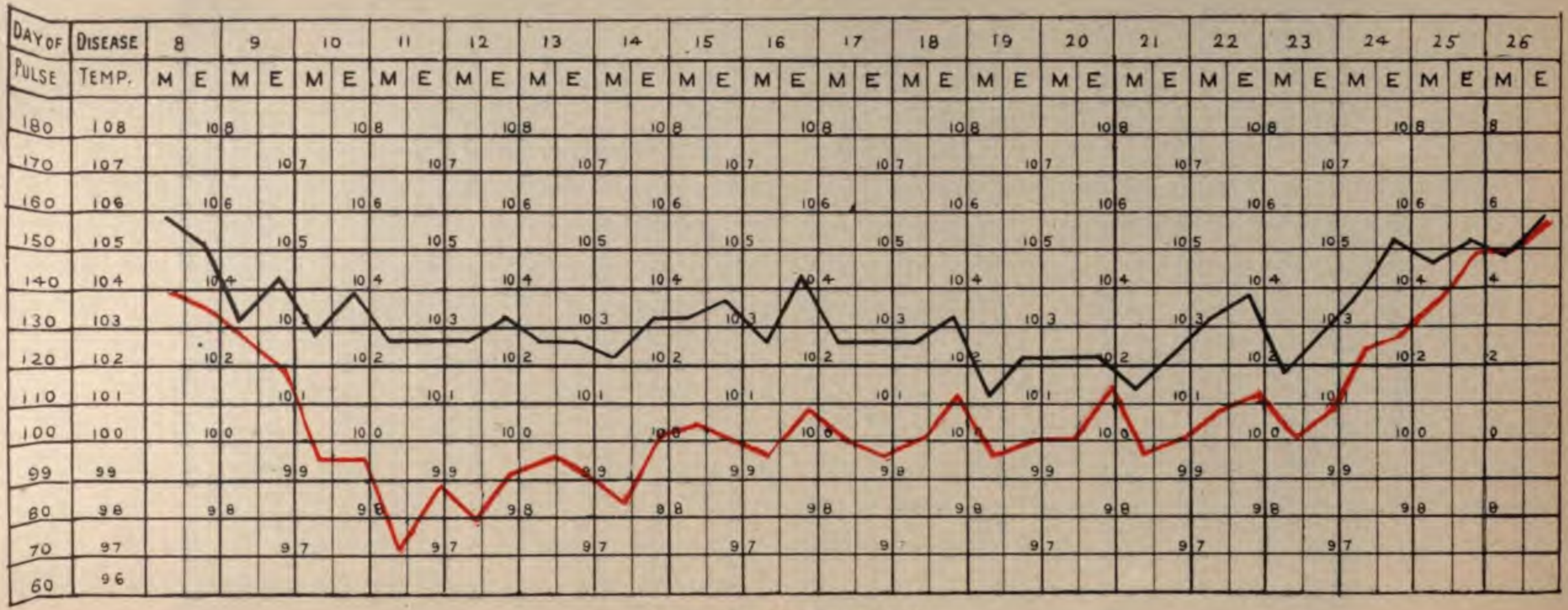
July 22: There was general hyperæsthesia, constant muscular twitchings, and muttering delirium.

July 29 he died.

(For chart of Case VI, see Fig. 17.)

Autopsy.—The inner surface of the dura mater is lined with a vascular membrane like that of chronic pachymeningitis, but thicker than usual. It is infiltrated with small blood clots of brownish color. One of the veins leading into the longitudinal

FIGURE 17.



Temperature black; pulse red.

sinus contains a large, white, puriform thrombus. The pia mater is congested, the brain tissue is normal.

The same membrane lines the dura mater of the spinal cord.

In the lungs all the bronchi contain pus and there are areas of broncho-pneumonia in the lower lobes.

The other viscera are normal.

THE PHARYNX AND TONSILS.

The nose, the pharynx, and the mouth are lined with a continuous tract of mucous membrane, which, on account of its situation, is exposed to a great variety of irritants, and is very often the seat of acute and chronic inflammations. The chronic inflammations are best treated by the specialist; the acute inflammations are usually left in the hands of the general practitioner.

Throughout this whole tract the structure of the mucous membrane is essentially the same—an outer covering of epithelium, a vascular stroma, a large supply of mucous glands.

On each side of the pharynx is a lymphatic gland of some size —the tonsil. The shape of the tonsils is peculiar; it is that of a flat mass of lymphatic glandular tissue folded on itself so as to form a wrinkled ovoid body, of which the outer surface is covered with a layer of epithelium. The wrinkles are the crypts of the tonsils.

We will consider:
Acute catarrhal pharyngitis.
Acute tonsillitis.
Acute croupous tonsillitis.
Suppurative tonsillitis.

ACUTE CATARRHAL PHARYNGITIS.

This is a simple, acute, exudative inflammation which involves circumscribed portions of the pharynx, or the entire throat with the tonsils, or extends also into the nose.

Lesions.—The mucous membrane is congested and swollen. The function of the mucous glands is at first arrested, so that the surface of the mucous membrane is dry; after some hours or days the function of these glands is stimulated. There is then an increased quantity of mucus, which is thin and easily discharged from the surface of the mucous membrane, or thick and tenacious so that it adheres to it.

Causes.—Some individuals and the members of some families are especially liable to acute pharyngitis; this liability is most marked in young people. The inhalation of irritating gases, or of irritating substances floating in the air, is a frequent cause of pharyngitis. Exposure to cold and wet, disturbances of digestion, and the poison of rheumatism are also efficient causes. It regularly occurs with the exanthemata and with typhoid fever.

Symptoms.—The symptoms are constitutional and local. The principal constitutional symptom is a rise of temperature from 100° to 105° F., with the accompanying chills, prostration, vomiting, headache, and pains in the limbs. This fever often precedes the sore throat and disappears before the latter has subsided.

The local symptoms are the characteristic appearance of the throat, and the pain, which is increased by any movement of the muscles of the pharynx. The pain is most annoying when the inflammation involves the pillars of the fauces, the upper surface of the soft palate, and the uvula. Children often do not complain of any pain in the throat. The quantity of mucus coating the posterior surface of the pharynx is sometimes so large as to look like a false membrane.

The cases vary as to the height of the temperature and the severity of the constitutional symptoms. In the worst cases with high temperatures the patients look seriously ill. The inflammation is a self-limited one, lasting for from four to seven days, but occasionally it continues for a longer time.

Treatment.—The best local application is a solution of ten grains of chloride of zinc in one ounce of a four per cent solution of cocaine, put on the throat with a brush. The best internal medication is five grains of salicylate of soda with one minim of tincture of aconite, given at first every hour, later at longer intervals.

ACUTE TONSILLITIS.

Synonyms.—Follicular tonsillitis, ulcerative tonsillitis, spotted sore throat.

Lesions.—There is a simple exudative inflammation of one or both tonsils. These bodies are swollen so that they can be seen projecting into the throat, and are congested. The crypts are filled with little white masses of mucus and epithelium, which contrast with the congested tonsils. There are no real ulcers.

The causes, symptoms, and treatment of acute tonsillitis are the same as those of acute pharyngitis.

CROUPOUS TONSILLITIS.

Synonyms.—Diphtheritic sore throat, false diphtheria.

Lesions.—The tonsils are congested and swollen. Their surfaces are partly or completely covered with patches of false membrane. The false membrane is composed of fibrin, pus, and necrotic epithelium. The stroma of the mucous membrane is infiltrated with fibrin and pus. The lymphatic glands in the neck may be swollen.

In bad cases there is gangrene of portions of the tonsils and of the tissues of the neck.

Streptococci and staphylococci are found in the inflamed tissues and the false membrane.

Causes.—This form of tonsillitis is due to local infection with streptococci and staphylococci. It is a complication of many of the infectious diseases, but especially of scarlet fever. Less frequently it occurs by itself.

Symptoms.—A croupous tonsillitis gives the same symptoms as does a simple tonsillitis, but much more severe.

The temperature is higher, the prostration greater, the appearance of the inflamed tonsils is quite different. The inflammation regularly runs its course within a week, but the prostration left after the disease is more marked and lasts longer.

The gangrenous forms are attended with septic symptoms and are usually fatal.

Treatment.—The most satisfactory treatment is the frequent and thorough irrigation of the throat with weak solutions of bichloride of mercury, peroxide of hydrogen, or formalin.

SUPPURATIVE TONSILLITIS.

Synonym.—Quinsy sore throat.

Lesions.—There is in one or both tonsils an acute exudative inflammation with neurosis, which goes on to the formation of an abscess in the substance of the tonsil. A catarrhal pharyngitis accompanies the tonsillitis.

Causes.—There is a decided predisposition in some persons and in some families to this form of tonsillitis. Apparently the exciting causes are the same as for simple tonsillitis, with the additional infection of the bacteria of suppuration.

Symptoms.—There is a febrile movement, which precedes and accompanies the tonsillitis. Accompanying the fever are chills, headache, vomiting, pains in the back, and prostration.

The inflamed tonsil gradually becomes more and more swollen; it fills up the pharynx, pushes the soft palate forward, and projects outward into the neck. From the beginning there is a good deal of pain in the throat, and as the swelling increases the pain is greater, the mouth and throat are constantly filled with mucus and saliva. The patients can hardly swallow anything; they feel as if they might suffocate; they are unable to sleep. If both tonsils are inflamed and the uvula swollen, or if there is œdema of the glottis, there is real danger of suffocation. All the symptoms continue and increase until the abscess breaks and the pus is discharged. Then there is an abrupt change for the better and in a few days the patient is well.

The inflammation regularly runs its course and terminates in rupture of the abscess within a week. If both tonsils are inflamed successively the disease lasts two or three weeks.

The patients almost uniformly recover, but it is possible for death to be produced by œdema of the glottis.

Treatment.—For the first two days the indications are to apply cold over the inflamed tonsil and to give aconite and the salicylate of soda internally. After this it is better to apply heat over the tonsil and to give enough opium to keep the patient comfortable. As soon as the abscess can be made out it should be opened. Swelling of the uvula and œdema of the glottis demand free scarification.

THE LARYNX.

Laryngismus Stridulus.

This name is given to two different forms of spasmodic contraction of the muscles of the larynx.

1. There is a spasmodic closure of the glottis, which is almost complete, lasts for a few seconds, and is then followed by a loud, stridulous inspiration.

This occurs in young children, usually under two years of age, who are badly nourished or who suffer from rickets.

It occurs in attacks, which are repeated after short or long intervals.

The child suddenly stops breathing, the face and lips become pale or slightly livid; the appearance is almost that of a dead person. Then, after a few seconds, there is a long, stridulous inspiration, and the attack is over. Sometimes the attacks are accompanied by rigidity of the muscles, or by general convulsions, or by unconsciousness. Occasionally the attacks are fatal.

At the time of an attack, if it is an alarming one, we may give inhalations of nitrite of amyl or pass a tube through the larynx.

To prevent the attacks the nutrition of the child is to be improved in every possible way.

2. There is a spasmodic partial closure of the glottis, attended with difficult and stridulous breathing, which lasts for several hours.

This occurs in young children of all kinds, whether their previous health has been good or not. There is a decided predisposition in families and in individuals. Some of the attacks are due to indigestion; for others no cause can be discovered.

The attacks usually begin in the night and last until the next day. The child wakes up in the night with laryngeal dyspnœa, which after a time is followed by more or less venous congestion of the skin. But there is no fever, and, except for the dyspnœa, the child does not look ill. Although the dyspnœa may appear alarming, it always eventually subsides, even if it is left to itself, but an emetic will cause it to disappear more rapidly. The best emetics are those which act the quickest; the yellow sulphate of mercury, apomorphia, ipecac, and antimony are those ordinarily used.

ACUTE CATARRHAL LARYNGITIS.

Lesions.—The mucous membrane of the larynx is congested, and at first dry. The stage of dryness lasts for from twelve to forty-eight hours, and is then succeeded by an increased production of mucus. With the production of the mucus the congestion and swelling of the mucous membrane diminish. The inflammation may extend to the trachea and bronchi or to the pharynx. It occurs both in adults and children.

1. IN ADULTS. *Causes.*—The inflammation occurs without discoverable cause, after exposure to cold, from the inhalation of smoke or steam, with syphilis, phthisis, measles, scarlatina, variola, erysipelas, typhus and typhoid fevers.

Symptoms.—In the more severe cases there is a febrile movement; in the milder cases this is absent.

The patients have a laryngeal cough, at first dry, afterward with mucous expectoration. The voice is husky, or stridulous, or reduced to a whisper. There is more or less discomfort or pain in the larynx. In some of the cases there is laryngeal dyspnœa, continuous, but with exacerbations. The most alarming feature about this dyspnœa is that occasionally the patients stop breathing suddenly and die.

The inflammation usually runs its course within two weeks, but it may be protracted for a much longer period.

Treatment.—During the acute stages of the laryngitis the continuous application of hot sponges to the neck, and the inhalation of steam, are of decided service. The internal administration of tartarized antimony or of the iodide of potash may also be useful. When the dyspnœa is urgent the patients are to be constantly watched, in order that intubation or tracheotomy may be performed, if necessary. If the inflammation is prolonged the mineral acids, quinine, iron, and change of climate may hasten the subsidence of the disease. The local application of astringent sprays is of much service.

2. In Children.—The catarrhal laryngitis of children is often called catarrhal croup. It is one of the most common diseases of childhood.

It occurs often without discoverable cause, sometimes after exposure to cold and wet, from the inhalation of steam and smoke, and as a complication of measles and scarlatina.

The disease is most common in children between the ages of one and five years, less frequent in older children. There is a well-marked predisposition to the disease in some children and in some families.

The changes in the mucous membrane of the larynx are the same as those observed in adults, but the smaller size of the larynx in children causes the swollen mucous membrane to be a still greater obstruction to the act of breathing.

Symptoms.—The local symptoms are: dyspnœa, which is continuous, but with exacerbations; in some cases attacks of laryngismus stridulus; stridulous voice or loss of voice; and stridulous cough.

The general symptom is a rise of temperature with its accompanying disturbances. The fever may precede, or follow, or be simultaneous with, the local symptoms.

The invasion of the disease is often sudden, and then usually occurs in the night, with the continuous dyspnœa or an attack of laryngismus stridulus as the first symptom.

In other cases the invasion of the disease is gradual, with a croupy cough as the first symptom.

The ordinary cases last for three days and nights, the symptoms worse during the night and better during the day.

The patients vary as to the height of the temperature and the degree of the dyspnœa. Most of the patients are decidedly better by the fourth day, the improvement beginning with the production of mucus from the inflamed membranes. Occasionally, however, the laryngitis is protracted for one or two weeks, or it may be succeeded by a bronchitis.

The Prognosis is good; even the severe cases are seldom fatal.

Treatment.—For the laryngitis we apply moist heat to the neck, and give internally five- or ten-drop doses of the wine of antimony every one or two hours. For the attacks of laryngismus stridulus we give emetics. For the fever it may be proper to give small doses of aconite or of acetanilid. The dyspnœa may be so great as to render intubation necessary. But to judge of this we must always listen to the lungs and see how well the act of breathing is carried on. The dyspnœa may look worse than it really is.

THE PLEURA.

"The pleuræ are serous membranes forming two shut sacs, quite distinct from each other, which line the right and left sides of the thoracic cavity, form by their approximation in the middle line the mediastinal partition, and are reflected each upon the root and over the entire free surface of the corresponding lung. Each pleura consists of a visceral and a parietal portion. The visceral portion, pleura pulmonalis, covers the lung; and the parietal portion lines the ribs and intercostal spaces (pleura costalis), covers the upper convex surface of the diaphragm, enters into the formation of the mediastinum, and adheres to the sides of the pericardium.

"The upper part of the pleura, together with the apex of the corresponding lung, rises into the root of the neck, reaching an inch, or even an inch and a half, above the first rib. Anteriorly the pleural sacs of opposite sides come nearly into contact behind the second piece of the sternum and continue so for some distance; but opposite the lower end of the sternum the right pleura passes beyond the middle line or remains close to it, while the left recedes to a variable distance. In the axillary line the right pleura extends down to the lower edge of the ninth rib, while the left pleura reaches to the lower edge of the tenth" (Quain's "Anatomy").

The structure of the pleura is that of a connective-tissue membrane. Its free surface is covered with a continuous layer of endothelial cells. The membrane itself is made up of fibres of connective tissue, reinforced by elastic anastomosing fibres. Running in parallel rows at the edges of the fibres are the fixed connective-tissue cells. These cells have large bodies with branching processes; they are most numerous in the layers of the pleura just beneath the endothelium. The blood vessels of the pulmonary pleura are continuous with those of the lungs; those of the costal pleura, with the vessels of the wall of the chest. In the costal pleura covering the intercostal spaces there is a very abundant system of lymphatics. It is convenient to think of the pulmonary pleura as a part of the lung which regularly shares in the inflammations which affect that organ. The costal, diaphragmatic, and mediastinal pleuræ, on the other hand, often become inflamed quite independently of changes in the lungs.

PLEURISY.

The different forms of pleurisy may be classified according to their causes, their clinical symptoms, or their anatomical changes. It is convenient to describe:

Pleurisy with the production of fibrin.
Pleurisy with the production of fibrin and serum.
Pleurisy with the production of fibrin, serum, and pus.
Pleurisy with adhesions.
Tubercular pleurisy.

In pleurisy with the production of fibrin alone, or of both fibrin and serum, the morbid changes in the pleura are essentially the same.

First, the production of fibrin and a few pus cells, either with or without serum.

Second, a gradual absorption of the serum and a disappearance of the fibrin.

Lastly, as the regular termination of the inflammation, the formation of permanent, new connective tissue in the shape of adhesions or of thickenings of the pleura. Variations from the regular course of the inflammation are effected by the excessive formation either of the fibrin, the pus, or the serum; and by the failure of the absorption of these inflammatory products.

If we endeavor to follow out the successive changes by which the fibrin, pus, and serum make their appearance and then disappear, and the way in which permanent new tissue takes their place, we encounter several difficulties.

It is impossible in any ordinary experience to obtain autopsies which will give the lesions belonging to each successive day of the disease; the pleura does not really show well if the patient has been dead more than two or three hours before the autopsy; in most cases the inflammation is too energetic, its products are too abundant, to be easily studied.

To obviate these difficulties we must resort to the lower animals. By injecting a saturated solution of chloride of zinc, with a hypodermic syringe, into the pleural cavity of a dog, we can excite a pleurisy exactly resembling that which we see in the human subject. By varying the amount of fluid injected, and by injecting it into one or both pleural cavities, we can obtain pleurisies of different degrees of intensity, and with different quantities of products of inflammation. By using a number of animals we can observe the course of the inflammation from hour to hour and from day to day.

The very first change we observe in such an artificial pleurisy is a simple congestion. The parietal pleura is of a uniform, rosy red color; its surface is shining, and moist as in health. There is no serum and no fibrin. Minute examination shows that the endothelial cells have fallen off in patches, and that where they have thus fallen off the superficial, connective-tissue cells are swollen and multiplied, and there are some pus cells. This is all that takes place for from half an hour to six hours after the injection has been made.

The next step in the inflammatory process is the appearance of serum and fibrin in variable amounts. The serum collects in the bottom of the pleural cavity. It is usually slightly turbid from the admixture of red blood cells and pus cells. The fibrin coats the whole surface of the pleura. There may be so little of it that it cannot be seen with the naked eye, or the quantity may be considerable.

The surface of the pleura loses its natural, glistening appearance in proportion to the quantity of fibrin coating it. The fibrin appears first in the form of little knobs and strings, or of a thin layer of granular matter. Where the endothelium still adheres the edges of the cells are separated from each other by granular matter. A few pus cells are seen entangled in the fibrin and embedded in the pleura. The swelling and new growth of the fixed connective-tissue cells is now very marked. The branching bodies of the old cells are readily seen. The new cells are of different shape from the old ones. They are polygonal cells, with a distinct body and nucleus, and are arranged in rows parallel with the course of the connective-tissue fibres. These new cells give the impression of being formed in some way from the old fixed cells, but precisely how I am unable to say. The time varies from half an hour to seven hours before these changes take place. By the end of twenty-four hours they are always fully developed.

On the second day of the pleurisy the gross and minute appearances are unchanged; only the layer of fibrin is thicker and there is more serum. Up to this time the lymphatic vessels and spaces can still be demonstrated with nitrate of silver, and are empty.

On the third day in some cases, not until the fourth in others, the growth of new cells has become well marked. The layer of fibrin on the pleura is more consistent; the serum is still abundant or has begun to disappear. The new cells present a great variety of shapes. They are abundant in the superficial layers of the pleura, but not in the deeper layers. They are also present in large numbers, entangled in the layer of fibrin coating the pleura, and in the adhesions of fibrin between the pulmonary and costal pleuræ.

By the fourth or fifth day new blood vessels make their appearance in the layer of fibrin and cells. These vessels can be injected from the aorta; they are continuous with the old vessels of the pleura. At the same time the fibrin begins to disappear, and the new cells form a more continuous layer.

By the sixth day the layer of fibrin and new tissue on the surface of the pleura is more adherent. It begins to form a part of the pleura.

The serum has by this time disappeared, and the quantity of fibrin is less. The new cells are still numerous, and they are becoming arranged into two divisions—a superficial layer, resembling the old endothelium, and a deeper layer of fusiform and branching cells.

After this time the quantity of fibrin continues to diminish, until by the fourteenth day it has entirely disappeared. Of the new cells, the superficial layer gradually forms a continuous layer of endothelium; in the deeper layer the rounded and polygonal cells are not as numerous, while there are many large and small fusiform and branching cells. Gradually a basement substance makes its appearance between the cells, at first homogeneous, afterward fibrillated.

These changes continue, until by the end of three weeks the pleura has its normal appearance, except that it is thickened and that there are adhesions between the costal and pulmonary pleuræ.

The lesions of human pleurisy are substantially the same as those just described. The only difference is that the fibrin, pus, and serum are produced in much larger quantities, and, as a result of this, the absorption of these products and the formation of new tissue follow more slowly.

We may then draw the following conclusions:

1. In pleurisy with the production of fibrin alone, or of fibrin and serum, two distinct pathological processes regularly take place. The blood vessels become congested, and through their walls transude the plasma of the blood and a few white blood cells. Part of the plasma appears as serum; part of it contains substances which are transformed into fibrin. At the very commencement of the inflammation the superficial, fixed connective-tissue cells also begin to increase in size and number.

2. The products of the first of these processes, the fibrin and serum, are regularly reabsorbed. In many patients, however, the course of the disease is irregular, and these products remain for a long time.

3. The product of the second of these processes regularly increases. The new cells, formed in the superficial layers of the pleura, wander into the fibrin, coating the pleura and forming adhesions, and there grow and multiply. New blood vessels and a new basement substance are formed between these cells.

4. The natural termination of such a pleurisy is the absorption of the fibrin and serum and the formation of new tissue coating the pleura and forming adhesions.

PLEURISY WITH THE PRODUCTION OF FIBRIN. DRY PLEURISY.

Lesions.—The inflammation may begin in the pulmonary, costal, or diaphragmatic pleura, according to the cause which produces it. The pleurisies beginning in the pulmonary pleura are always secondary to changes in the lungs. Those, on the other hand, which begin in the costal pleura are often quite independent of any inflammation of the lungs. Usually only a circumscribed portion of the pulmonary, costal, mediastinal, or diaphragmatic pleura is involved, but sometimes the entire pleura of one side of the chest is inflamed. The inflammation regularly extends to the portion of pleura opposite to it. The inflamed pleura is coated with fibrin, and bands of fibrin form adhesions between the opposed pleural surfaces. As a rule the layer of fibrin is thin, but there are rare cases in which the quantity of fibrin is enormous and the lung is compressed by it as if by fluid.

When the inflammation has subsided the fibrin is absorbed, but permanent connective-tissue thickenings and adhesions are left.

Causes.—The pleurisies which originate in the pulmonary pleura are caused by changes in the lungs. Lobar pneumonia is nearly always accompanied by dry pleurisy, broncho-pneumonia less often. The tubercular inflammations of the lungs, both acute and chronic, are frequent causes of dry pleurisy, and this is the case even when the tubercular inflammation of the lungs is of small area and does not extend directly up to the pleura. Often, indeed, a dry pleurisy is the first thing to tell us that there is a tubercular inflammation of the lung.

The inflammations which begin in the costal pleura, on the other hand, are seldom caused by tuberculosis. Most of them occur during the cold and damp seasons of the year and are apparently caused by exposure. Some persons evidently have an individual predisposition to this form of pleurisy and suffer from a number of attacks. We also see dry pleurisy with wounds of the chest wall, with peritonitis, with rheumatism, and with the infectious diseases, especially with influenza.

As most persons recover from primary dry pleurisy, but little is known about its bacteriology.

Primary dry pleurisy is a disease of ordinary occurrence in New York; every winter we see many cases in all classes of society.

Symptoms.—The most constant physical sign is a friction sound. The sound is of a rubbing or grazing quality, or it has the character of a crepitant, subcrepitant, or mucous râle. The friction sound is heard directly over the inflamed portion of the pleura. It cannot be heard if only the diaphragmatic or mediastinal pleura is inflamed. It is heard only with inspiration, or with expiration also; it sounds as if it were close under the ear. It can be heard best after deep breathing or coughing.

In some cases there is also dulness over the inflamed portion of the pleura.

In a dry pleurisy accompanying lobar and broncho-pneumonia the friction sound is heard when the inflammation reaches the surface of the lung. It has the characters of a crepitant or subcrepitant râle. The pleurisy seems to be responsible for the pain from which these patients suffer.

With acute or subacute miliary tuberculosis of the lungs a dry pleurisy may either mark the invasion of the tubercular inflammation of the lungs or it may be repeated from time to time as the tuberculosis goes on. A friction sound, pain, and a rise of temperature regularly accompany these attacks.

Whenever a patient has the ordinary symptoms of dry pleurisy, and after ten days, with a clean tongue, a fair appetite, and no great feeling of illness, there is a regular rise of temperature every afternoon, it is probable that the pleurisy marks the invasion of a miliary tuberculosis of the lungs.

The dry pleurisy which accompanies the acute tubercular inflammations of the lung which cause consolidation behaves like the dry pleurisy of broncho-pneumonia.

Epidemic influenza furnishes us with a considerable number of cases of dry pleurisy, and the inflammation of the pleura may be developed at any time in the course of the influenza. Not infrequently the whole of the pleura on one side of the chest is involved.

Of the primary dry pleurisies involving the costal pleura, the cases vary as to the extent of pleura involved. With inflammation of a small portion of the pleura the friction sound is circumscribed, the pain is not great, the temperature is not high or remains normal, the patient is not confined to bed, the pleurisy runs its course within a week. With more extensive pleurisies the friction sound can be heard over the whole of the posterior wall of the chest, the pain is more severe, the temperature is higher, the patient may be confined to bed for one or two weeks. In some of the cases there is a dry cough.

While the pain is regularly referred to the position of the inflamed pleura, exceptions to this rule are not uncommon. Many patients refer the pain to the lumbar region (it may be confounded with the pain of lumbago or of renal colic); some refer it to the abdomen and some to the opposite side of the chest. The pain of dry pleurisy is, as a rule, less severe than that of an acute pleurisy with effusion.

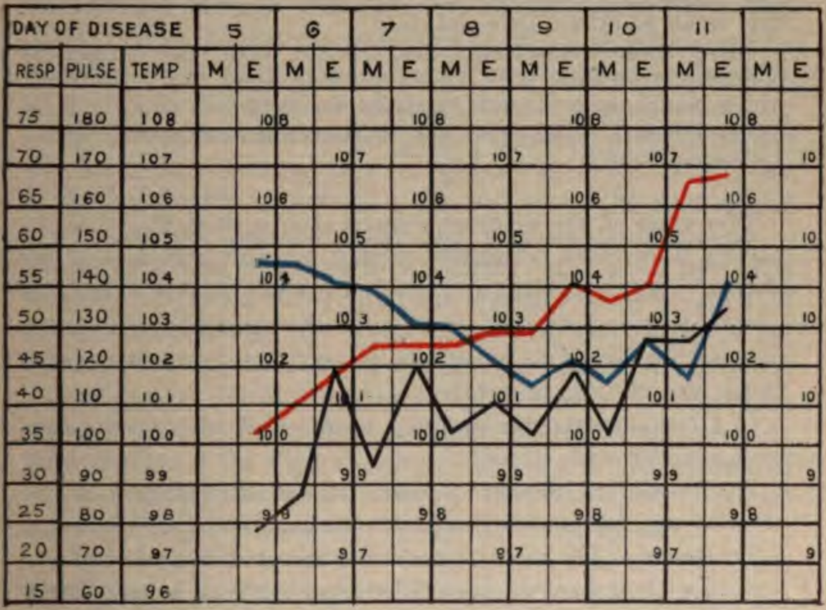
A dry pleurisy confined to the upper surface of the diaphragm may simulate an acute peritonitis. There are pain and tenderness over the upper part of the abdomen, with a febrile movement and considerable prostration, while no friction sound can be heard.

There are cases of pleurisy with a fibrino-purulent exudation which behave very differently from ordinary cases of dry pleurisy. They seem to be due to infection with streptococci or pneumococci. The inflammation involves one pleura, or both pleuræ, or both pleuræ and the pericardium. The inflamed membranes are coated with a layer of fibrin infiltrated with leucocytes. The symptoms resemble those of lobar pneumonia or of sepsis. The physical signs are only those of dry pleurisy. The diagnosis is extremely difficult. These cases are apt to end fatally.

Case I.—Male, 38 years old, December 21, 1898. Four days ago, in the evening, was taken suddenly with a chill followed by fever, marked prostration, cough with scanty mucous expectoration and a sharp pain in the right side, made worse by cough and inspiration. These symptoms have continued up to the present time.

Now, on the fifth day, prostration is marked, the breathing is not labored, urine 1022, a considerable quantity of albumin. Over the whole right lung, in front and behind, there are crepitant and subcrepitant friction sounds. Over the right lung behind, from the angle of the scapula down, there is dulness on percussion. The heart's action is rapid and regular; the apex is in the fifth space, just inside the nipple; there is no murmur.

FIGURE 18.



Temperature black; pulse red; respiration blue.

Ninth day: The man has been gradually losing, the breathing is rapid and shallow, he is becoming delirious. He continued in this condition until he died on the eleventh day.

(For chart of Case I, see Fig. 18.)

Autopsy.—The entire right lung and the lower lobe of the left lung are coated with a thick layer of fibrin; the bronchi contain muco-pus; there is no exudation in the lungs themselves. The pericardium is coated with a thin layer of fibrin.

The kidneys show the lesions of acute degeneration.

The cases with very large quantities of fibrin are of rare occurrence. The following is an example:

CASE II.—A man, 39 years old, always strong and healthy, on May 4 fell into the river. The next day he was attacked with symptoms of pneumonia. I saw him first on the fourteenth day of his disease. At that time there were the physical signs of consolidation of the entire left lung, with subcrepitant râles at the base of the right lung. The temperature was 105° F., the breathing 46, the pulse 120. He continued with the same physical signs, and temperatures between 102° and 104° F., until the twentieth day, when he died rather suddenly.

After death it was found that the left costal and pulmonary pleuræ were coated with an enormously thick layer of fibrin, which compressed the lung against the spine, as if by a large serous effusion. The lower lobe of the right lung was congested and œdematous.

Most cases of dry pleurisy recover after a short time, but the patient is left with permanent thickenings and adhesions of the pleura. Such adhesions may give no further trouble, except for occasional pain; or they may form the starting point for a chronic pleurisy with adhesions, followed by interstitial pneumonia and chronic bronchitis.

In a few cases the dry pleurisy is succeeded after several days by a pleurisy with effusion.

In a moderate number of cases one or more attacks of dry pleurisy are followed by tubercular inflammation of the lungs.

Treatment.—The more severe cases are glad to go to bed and stay there, but many of the milder cases continue at their work, do not feel sick, and are unwilling to be confined to the house. It seems prudent, however, to keep even the milder cases in the house until the friction sound has disappeared. The pain can sometimes be relieved by strapping the chest with adhesive plaster; in other cases it is necessary to use opium.

The pleurisy really runs its own course and takes care of itself, but it is customary to employ dry cups or blisters over the inflamed pleura.

PLEURISY WITH THE PRODUCTION OF FIBRIN AND SERUM. PLEURISY WITH EFFUSION.

Lesions.—The inflammation is, as a rule, not circumscribed, but involves the greater part of the costal and pulmonary pleuræ on one side of the chest.

Sometimes, however, the extent of the inflamed pleura is but small, and the inflammatory products are shut in by adhesions. Rarely the pleuræ of both sides of the chest are inflamed, and when this is the case there is apt to be also pericarditis. This is also true of the pleurisy which can be produced experimentally in animals. If we irritate the pleuræ on both sides of the chest, in addition to the double pleurisy there will be pericarditis.

The surface of the inflamed pleura is coated with fibrin, and bands of fibrin join together its opposed surfaces. The serum is clear or turbid; it contains a few leucocytes. Red blood cells are not usually present, unless the pleurisy is tubercular or caused by a new growth or by an injury. But there are a few cases which do not differ from the ordinary cases, except that the serum is bloody. As a rule no bacteria are present in the serum. The fluid collects in the most dependent part of the pleural cavity, unless it is shut in by adhesions. The quantity varies from a few ounces to a quantity so large as to completely fill and distend the pleural cavity. The degree of compression of the lung varies with the quantity of the fluid. In the extreme and long-continued cases the lung is almost unaërated.

After the inflammation has subsided the serum and fibrin are absorbed, and thickenings and adhesions of connective tissue are left.

The compressed lung expands partially or completely. According to the expansion of the lung there is left more or less retraction of the affected side of the chest.

Causes.—The pleurisies with effusion, like the dry pleurisies, originate in the pulmonary, costal, or diaphragmatic pleura. Lobar pneumonia, broncho-pneumonia, and the tubercular inflammations of the lungs are often accompanied by pleurisy with effusion. The poisons of rheumatism, of influenza, and of the infectious diseases are causes of pleurisy. Wounds of the walls of the chest, fracture of the ribs, and blows on the chest cause pleurisies with effusion, which are often hemorrhagic. Abscesses of the right lobe of the liver may cause effusion into the right pleura. The new growths of the pleura are regularly attended with large effusions of serum into the pleural cavity.

Pleurisies with effusion which exactly resemble the ordinary pleurisies of human beings can be produced by injecting chloride of zinc into the pleural cavities of dogs.

Of late years it has become the fashion to regard nearly all the primary cases of pleurisy with effusion as of tubercular origin. I must confess that this view seems to me erroneous. There is a true primary tubercular inflammation of the pleura which it is important to recognize. The prognosis and results of treatment are entirely different from those of ordinary pleurisy with effusion. The lesions, symptoms, and course of a pleurisy with effusion are those of a simple exudative inflammation.

The behavior of the acute cases is such as to make it probable that infection by the pneumococcus is the cause of the inflammation, and this belief has been confirmed in a considerable number of cases. But in a large number of cases the fluid is found to be sterile. The most difficult cases to account for are those which begin insidiously and run a subacute course.

Symptoms. Physical Signs.—At the beginning of the inflammation, when the pleura is coated with fibrin and but little serum has been exuded, there is a friction sound, which is a rubbing sound, or a crepitant or subcrepitant râle. After the fluid has been absorbed there is again a friction sound—a subcrepitant or coarse râle, or a creaking sound.

When a considerable quantity of fluid has accumulated in the pleural cavity there are physical signs due to the presence of the fluid and the compression of the lung.

Below the level of the fluid there is flatness on percussion, absence of voice, of breathing, and of vocal fremitus. The fluid accumulates in the lower part of the pleural cavity, compressing the lung upward and against the vertebral column; or in the posterior part of the pleural cavity, compressing the lung against the anterior wall of the chest; or it may be shut in by adhesions.

The compression of the lung is in proportion to the quantity of the fluid.

At the level of the fluid there is dulness on percussion and ægophony.

Above the level of the fluid, over the compressed lung, the percussion resonance is pulmonary, or exaggerated and high-pitched, or tympanitic. The breathing is pulmonary, or exaggerated, or broncho-vesicular, or bronchial.

The affected side measures more than the opposite side of the chest, the diaphragm is pushed down, the intercostal spaces may be forced outward, the heart may be displaced toward the opposite side of the chest.

As the fluid is absorbed and the voice and breathing can be heard lower and lower down, the flatness disappears, but dulness on percussion remains for some time after all the fluid has been absorbed.

Exceptional Physical Signs.—The vocal fremitus may not be lost below the level of the fluid.

Bronchophony alone, or both bronchial voice and bronchial breathing, may be heard below the level of the fluid, especially if the quantity of fluid is large and the lung compressed. These physical signs are so often present that it is hardly proper to call them exceptional.

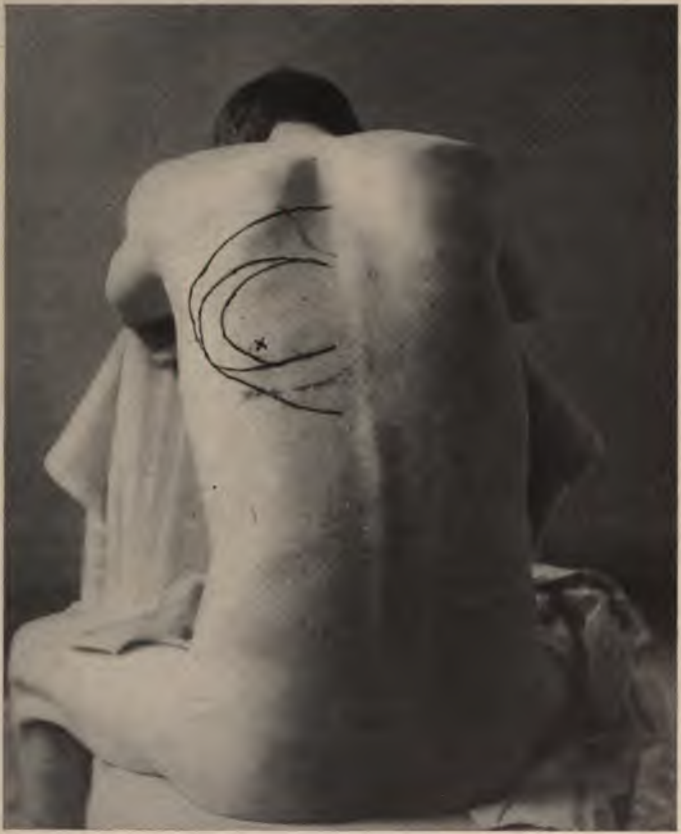
Above the level of the fluid, over the compressed lung, there may be cavernous breathing and gurgling râles. Walshe says that he has once heard the cracked-pot sound in the first and second interspaces during the height of effusion, and once a loud rubbing friction sound above the clavicle.

A subcrepitant râle may be heard below the level of the fluid, the level being demonstrated by the aspirator. This is often a very deceptive sign. It is difficult to believe that fluid is present when you hear a subcrepitant râle over it.

The sacculated effusions give irregular physical signs, which vary with the position of the fluid. The effusions are most easily made out if they are in contact with the wall of the chest.

Most of the sacculated pleurisies I have seen have been situated about the root of the lung, gave the maximum flatness at some point between the scapula and the vertebral column, and were best aspirated at the point of maximum flatness. In some of these cases there was flatness and absence of breathing over the fluid; in some, flatness with bronchial voice and breathing; in some there was pulmonary resonance with bronchial voice and breathing over the lower part of the chest where there was no fluid.

FIGURE 19.



Sacculated pleurisy with effusion. Over the inside half-circle, flatness on percussion, bronchial voice, and broncho-vesicular breathing. The cross marks the point of aspiration.

(See Fig. 19.)

Less frequently I have seen the fluid sacculated in the upper part of the chest.

The most troublesome cases to make out are those with several collections of fluid shut in in different parts of the chest.

The Course of the Disease.—From a clinical point of view it is convenient to distinguish three forms of pleurisy with effusion.

1. The acute form. The symptoms of the invasion and of the first two days are the same as those of a lobar pneumonia, and give one the impression of a pneumococcus infection which has attacked the pleura instead of the lung. The patients, often after a well-marked exposure, are attacked with chills, a rapid rise of temperature up to 103° or 104° F., full and frequent pulse, pains in the head and limbs, vomiting, and prostration. The breathing is frequent, shallow, and difficult. The patient is much distressed by the pain, which is increased by every movement of the body. The pain is referred to the inflamed pleura, or to some point in the back, or in the abdomen, or even to the opposite side of the chest. It usually becomes less severe with the accumulation of serum in the pleural cavity.

While the constitutional symptoms are going on the serum is being rapidly transuded. Within three days the pleural cavity is often half full, sometimes even distended by the fluid.

The febrile movement regularly runs its course and nearly subsides within two weeks. How long the fluid remains in the chest, with an afternoon fever, depends largely upon the treatment.

Although the patients are very seriously ill, yet, as a rule, they recover. But there are cases which terminate fatally, and some of these die very suddenly, without anything at the autopsy except the pleurisy.

CASE III.—Female, 22 years old, November 17, 1896. Yesterday morning had a chill; pain in the left chest, made worse by breathing and motion; fever, headache, and marked prostration.

To-day color and nutrition fair; tongue moist and clean; pain in the left side; no cough; marked prostration; urine 1010, no albumin or casts.

Over the left chest behind, from the angle of the scapula down, there is flatness on percussion, with absence of voice and breathing. The heart is normal.

Tenth day: Aspiration of twenty-four ounces of clear serum.

Twenty-fifth day: The patient is well.

(For chart of Case III, see Fig. 20.)

CASE IV. *Sudden Death.*—Male, 38 years old, March 10, 1887. For two weeks past has been sick with fever, cough, dyspnœa, and pain in left chest.

To-day: Pulse 140, breathing 48, temperature 103.6° F.; urine 1015, albumin and casts.

Left chest half full of fluid.

Fifteenth day: In the morning sat up a few minutes; went back to bed and at 10.25 A.M. suddenly died.

Autopsy (seven hours after death).—The left pleura was half full of turbid serum; the pleura was coated with fibrin; the lung was somewhat compressed, but still aërated, neither congested nor œdematous. The right lung was large, over-inflated, congested, with pus in the small bronchi.

The kidneys were of normal size, their surfaces smooth, the cortex thickened. The brain, heart, liver, spleen, stomach, and intestines showed no lesions.

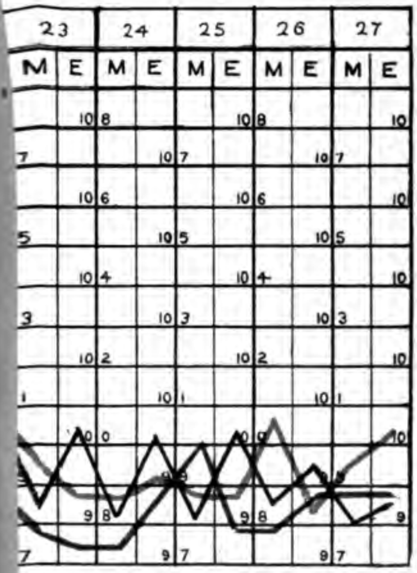
CASE V. *Sudden Death after Aspiration.*—Male, 70 years old, September 26, 1872. The man has had bronchitis for some time, but was able to work until one week ago. Then he was attacked with severe dyspnœa and sharp pain in the left side of the chest.

To-day: Marked dyspnœa, severe pain in left chest. Over the entire left chest flatness on percussion, absence of voice and breathing, bulging of intercostal spaces; the left side measured one inch more than the right. There is a mitral systolic murmur.

Tenth day: In the morning thirty-two ounces of clear serum were aspirated from the left chest. At 8 o'clock that evening he died suddenly.

Autopsy.—The left pleural cavity is partly filled with clear serum, the pleura is congested and coated with a thick layer of fibrin, the lung is compressed. The right lung is emphysematous; the lower lobe is congested and œdematous. The mitral valve is somewhat thickened and the left ventricle a little hypertrophied. Large white kidneys and a fatty liver.

2. There are cases of pleurisy with effusion in which the invasion is acute, and yet not violent, while the transudation of serum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is rather slow. The patients have a temperature of from 100° to 103° F., higher in the afternoon than in the morning. They feel ill, but do not always go to bed. They have more or less pain in the side, cough, and dyspnœa. If the patients are left to themselves the fluid in the chest may continue to increase for several weeks. In some of these patients there is a curious absence of pulmonary symptoms—no cough, no dyspnœa, no pain in the chest; very little to observe, except a febrile movement and the physical signs. While many of these patients do well with almost any treatment, others continue to have the pleurisy until the chest is efficiently aspirated.

CASE VI.—Male, 31 years old, February 19, 1885. Eleven days ago the man was attacked with pain in the left side, chill, fever, headache, dyspnœa, and a dry cough.

To-day: Pulse 94, temperature 101.8° F.; urine 1014, no albumin. Over the left lung behind, from the angle of the scapula down, there is dulness on percussion, absence of breathing and bronchial voice.

Eighteenth day: The temperature has continued between 100° and 102° F.; the fluid has increased in quantity, so that there is now flatness on percussion and absence of breathing over the whole left chest behind.

Seventy-eighth day: The man has continued with temperatures between 99° and 101° F.; the fluid has decreased, so that the chest is only half full of fluid.

A small syringeful of clear serum was drawn off on the forty-eighth day. He left the hospital with the chest still half full of fluid. He received only medical treatment and could probably have been easily cured by aspiration.

CASE VII. *Pleurisy with Effusion, Sacculated.*—Male, 47 years old, November 24, 1896. The man had pneumonia three years ago.

For five days he has had occasional chilly sensations, with cough, headache, and pain in the lower part of the chest behind. There has also been some nausea and vomiting.

Now he does not look very sick; the tongue is moist and coated; temperature 101.4°, pulse 112, breathing 28; urine 1028, no albumin or casts.

Over the left lung behind there is an area about eight inches in diameter over which there is bronchial voice and dulness. See Fig. 19.

Ninth day: Five hundred and fifty cubic centimetres were aspirated at the point shown in the photograph.

Eleventh day: The temperature was normal.

Twentieth day: The man was discharged cured.

3. There are cases in which the invasion is so slow and insidious that it is difficult to tell the precise date of the beginning of the inflammation. Many of the patients are sick for weeks or months before they seek medical advice. For a long time they are not confined to bed; they often continue at their ordinary occupations; some even do laborer's work. But the quantity of fluid in the chest slowly increases, the appetite fails, the patients lose flesh and strength and become anæmic. The pulmonary symptoms are: pain in the side, dyspnœa on exertion, and a dry cough. But it is not uncommon to see persons who complain of nothing but the loss of flesh and strength, and the diagnosis has to be made by the physical signs. An afternoon fever of from 100° F. to 103° F. is regularly present, sometimes followed by sweating at night.

It seems to be possible in any of these forms of pleurisy to have a later infection with streptococci and a change of the inflammation into a purulent one.

After recovery from pleurisy with effusion, there are left behind changes in the chest which are in proportion to the quantity of fluid and the length of time it remains in the pleural cavity.

The expansion of the lung is complete or incomplete. If incomplete the wall of the chest is retracted and the heart pulled to one side.

The adhesions which are left behind often give occasional pain for many months. They may cause chronic pleurisy with adhesions, or chronic bronchitis, or interstitial pneumonia.

In a moderate number of patients pleurisy with effusion is followed by tubercular inflammation of the lungs. But the interval between the two may be of months' or years' duration, and there is no evidence that the two are in any way related to each other.

The displaced heart may return to its natural position, it may remain displaced and fastened by adhesions, or it may be drawn toward the retracted side of the chest.

Case VIII. *Subacute Pleurisy.*—Male, 34 years old, November 21, 1888. For about a month the man has been losing appetite, flesh, and strength.

For three weeks he has had dry cough, pain in the left side of the chest, and dyspnœa, but continued at work until three days ago.

To-day: Pulse 108, breathing 36, temperature 102.6° F.; urine 1018, no albumin.

Over the left chest behind, from the angle of the scapula down, there is flatness on percussion, absence of vocal fremitus, bronchial voice, and bronchial breathing.

Thirty-eighth day: The fluid in the chest has increased in quantity; 2,100 c.c. of clear serum were drawn off.

Fifty-first day: There is no more fluid in the chest; the man is well.

Case IX. *Subacute Pleurisy; Inefficient Treatment.*—Female, 19 years old, December 22, 1886. Five months ago she began to have pain in the left side and headache.

For two months the pain in the left side has been more constant, there has been dyspnœa on exertion, and she has felt hot and thirsty.

Now she is a large, stout girl, somewhat anæmic, with no appetite; pulse 120, respiration 32, temperature 99° F.; urine 1030, no albumin or casts. Over the entire left chest there is flatness on percussion; behind there is bronchial voice and breathing; the heart is pushed to the right.

She was put on solid diet, one pint of milk a day, no other fluid; five grains of chloride of sodium three times a day.

December 24: Twenty-five ounces of clear serum were aspirated.

January 1: She is only passing twelve ounces of urine a day. She is now to take all the fluids she wants and the compound diuretic pill.

January 4 to 6: She is passing from ten to twelve ounces of urine a day.

January 6: She is given three grains of caffein four times a day.

January 27: She has been passing from twenty to thirty-five ounces of urine a day; the fluid in the chest has diminished; the caffein is stopped.

February 3: There is no more fluid in the chest.

In patients with small effusions in the bottom of the chest the diagnosis is sometimes difficult. The percussion note is dull rather than flat; the breathing can be heard, but the voice is usually diminished. It is hardly possible to be sure of them without the needle.

It is also to be noted that some persons have several attacks of pleurisy with effusion, first on one side, then on the other.

Treatment.—The ordinary treatment of pleurisy with effusion at the present time is to try some plan of medical treatment for two or three weeks, and if the effusion increases, or does not decrease sufficiently, then to resort to aspiration. Salicylate of soda, iodide of potash, and the diuretics are the drugs usually given; counter-irritation is employed over the chest, and sometimes the quantity of fluids ingested is very much restricted.

My own belief is that the best treatment for pleurisy with effusion is by the early aspiration of the fluid without the use of drugs.

When I began to practise medicine in 1866 the routine treatment in New York was to give potassium iodide internally and to apply repeated blisters over the affected side of the chest. When the chest was extremely distended the fluid was drawn off with a trocar and canula. The patients, as a rule, did not die, but they were sick for a long time. The extensive pleuritic adhesions and the long-continued compression of the lung left the patients liable to interstitial pneumonia, chronic bronchitis, and pulmonary tuberculosis.

As late as 1881 I taught as follows:

"In those cases in which the invasion of the disease is acute the treatment varies according to the rational symptoms. If the patient is strong and robust, as a rule considerable relief may be afforded by applying wet cups over the affected side of the thorax. If the patient is not strong and vigorous, dry cups or blisters may be used. To relieve the pain and diminish the fever opium, aconite, and liquor ammonii acetatis are given. After the acute symptoms have subsided the question of getting rid of the fluid in the thoracic cavity comes up.

"In those cases in which the invasion is insidious there are two indications to be met. In the first place the fluid should be removed from the pleural cavity, and in the second place the nutrition of the patient must be improved. The exact way in which these cases of subacute pleurisy are to be treated depends on the general condition of the patient. If his nutrition has not suffered up to the time he comes under treatment, your attention should be principally directed to the removal of the fluid by means of persistent irritation of the skin of the chest by blisters, and by the internal use of the iodide or acetate of potash. If, however, when the patient comes under observation, his nutrition is impaired and he is emaciated and anæmic, less attention should be paid to the fluid in the pleural cavity.

"The principal effort should be directed to the improvement of his general health. Such a patient will not bear blisters, nor can his condition be improved by the use of diuretics. Iron, quinine, and the mineral acids should be administered together with cod-liver oil or cream.

"It may be necessary to send the patient to a warm climate.

"Besides these means of treatment the question of mechanical removal of the fluid from the pleural cavity presents itself. In regard to the advisability of aspirating the thorax there is still a considerable difference of opinion in the profession. It is, however, generally agreed that when the dyspnœa is so great as to threaten the life of the patient, and when the fluid in the pleura does not diminish within a reasonable length of time, it is expedient to perform the operation. The reasons given for not aspirating are: That in a considerable number of cases a single aspiration is of very little service, for after the fluid has been drawn off it will reaccumulate and other aspirations are necessary; that with a number of aspirations there is danger of purulent infection; that a number of sudden deaths have occurred within twenty-four hours after aspiration."

Such a plan of treatment as is outlined above is still followed by many. It has been somewhat modified by the "thirst cure" and by the use of the salicylate of soda. The radical objection to this plan is the intolerable length of time during which the patient remains sick. Deaths, indeed, are few, but two months is an ordinary duration and six months is not uncommon.

Still further than this, the longer the pleurisy lasts and the longer the lung is compressed, the greater is the extent of the pleuritic adhesions and the greater the vulnerability of the lung to tubercular inflammation.

As time has gone on I have been so thoroughly impressed with the idea that aspiration is the only treatment needed for pleurisy with effusion that I have thought it worth while to tabulate two hundred ordinary hospital cases of pleurisy with effusion occurring from the year 1886 to 1901.

In the first 82 cases of the table, besides the aspiration, medical treatment was employed; in the subsequent cases aspiration was the only treatment.

It will be noted, in the first place, that none of the patients died, none of them were injured by the operation, and in none of them was the chest infected.

In all of the patients the quantity of fluid in the chest was considerable and occupied the lower part of the pleural cavity.

The fluid taken from the chest was bloody in 25 cases, turbid in 18 cases, and clear serum in the rest. The character of the fluid seemed to make no difference. Those with bloody or turbid serum did as well as those with clear serum.

In 121 of the cases the pleurisy was on the left side, in 79 on the right side.

The quantities of fluid aspirated were as follows: Six cases 3,000 to 3,500 c.c., twenty-three cases 1,500 to 2,000 c.c., fifty-five cases 1,000 to 1,500 c.c., forty-nine cases 500 to 1,000 c.c., thirty cases 100 to 400 c.c.

Of the 200 cases 182 left the hospital entirely cured; 6 left the hospital within ten days after the aspiration and probably recovered; 6 left the hospital at periods of from seventeen to thirty-six days after the aspiration—the pleurisy was better, but the results were uncertain; 6 left the hospital at the end of from twenty-four to thirty-eight days, not at all improved.

Of the 182 cases which were cured, the length of time which elapsed from the beginning of the pleurisy to the day of aspiration was as follows:

Seven months, one case.

Ninety days, five cases.

Seventy to seventy-five days, three cases.

Sixty to seventy days, nine cases.
Fifty to sixty days, six cases.
Forty to fifty days, nine cases.
Thirty to forty days, fifteen cases.
Twenty to thirty days, thirty-nine cases.
Ten to twenty days, seventy-one cases.
Five to ten days, twenty cases.
Four days, two cases.
Three days, one case.

It will be seen that in more than one-half the cases the duration of the pleurisy before aspiration was between ten and thirty days.

In all of the cases the pleurisy was going on as an inflammation up to the time of the aspirations; the patients had not been at all improved, either by nature or by the different medicines which they had taken.

The length of time which elapsed from the aspiration to the entire cure of the patient was as follows:

Within a week, 64 cases.
Within two weeks, 138 cases.
Within three weeks, 158 cases.
Within four weeks, 169 cases.
Within six weeks, 179 cases.

As to the number of aspirations: the operation was done once in 142 cases, twice in 45 cases, three times in 9 cases, and four times in 4 cases.

In private practice the results are better than in the hospital. In the fortunate cases within twenty-four hours after one aspiration there is no more fluid and no more pleurisy.

In a large number of cases the pleurisy is cured within a week, and none of them ought to be sick longer than two weeks.

Cases of Pleurisy with Effusion Treated by Aspiration at the Roosevelt Hospital, from 1886 to 1901.

No.	Age.	Condition of the chest on admission.	Day tapped.	Character of the fluid.	Subsequent tappings.	Other treatment.	No. of days from first tapping to end of pleurisy.
1	28	Left chest two-thirds full of fluid.	28th day	Reddish serum 1600 c.c.	None.	Acetate of potash and digitalis, calomel, squills, and digitalin; strapping the chest.	38 days.
2	45	Half full of fluid.	15th "	Clear serum 1400 c.c.	"	Calomel, squills, and digitalin.	8 "
3	36	Left chest two-thirds full of fluid.	120th "	Clear serum 1500 c.c.	"	None.	Went home on 9th day with chest still holding a little fluid.
4	30	Left chest three-fourths full of fluid.	60th "	Clear serum 2000 c.c.	"	Digitalin.	14 days.
5	34	Left chest three-fourths full of fluid.	16th "	Clear serum 3000 c.c.	"	None.	14 "
6	18	Left chest full of fluid.	60th "	Clear serum 1800 c.c.	One 400 c.c.	"	24 "
7	46	Right chest half full of fluid.	35th "	Clear serum three pints.	One 975 c.c.	"	13 "
8	33	Right chest three-fourths full of fluid.	9th "	Dark serum 3000 c.c.	None.	"	14 "
9	57	Left chest two-thirds full of fluid.	21st "	Clear serum 1900 c.c	"	Digitalin.	7 "
10	23	Left chest half full of fluid.	14th "	Clear serum 1000 c.c.	"	"	6 "
11	27	Left chest full of fluid.	35th "	Reddish ser. 3150 c.c.	"	"	20 "
12	22	Right chest full of fluid.	90th "	Clear serum 24 ounces.	"	"	19 days; improved.
13	31	Left chest full of fluid.	12th "	Dark red ser. 3875 c.c.	"	"	30 days.
14	51	Right chest half full of fluid.	8th "	Clear serum 2100 c.c.	"	Potassium iodide.	14 "
15	32	Left chest half full of fluid.	14th "	Clear serum 825 c.c.	"	None.	6 "
16	25	Right chest half full of fluid.	30th "	Clear serum 1750 c.c.	"	Sodium salicylate; strapping the chest	5 "
17	8	Right chest two-thirds full of fluid.	14th "	Reddish ser. 4 ounces.	"	None.	16 "
18	23	Right chest two-thirds full of fluid.	12th "	Clear serum 2400 c.c.	"	Digitalin, diuretic pills.	4 "
19	31	Right chest half full of fluid.	14th "	Clear serum 1050 c.c.	Bloody ser. 200 c.c.	Digitalis, squills, strychnia; strapping the chest.	26 "
20	28	Left chest half full of fluid.	42d "	Clear serum 300 c.c.	None.	Digitalin.	7 "
21	33	Left chest full of fluid.	28th "	Brown serum 3200 c.c.	"	None.	16 "
22	25	Left chest half full of fluid.	14th "	Clear serum 2000 c.c.	"	"	10 "
23	50	Right chest half full of fluid.	28th "	Clear serum 750 c.c.	800 c.c. 90 c.c. 575 c.c.	Digitalin.	43 "
24	42	Left chest full of fluid.	14th "	Clear serum 2000 c.c.	None.	None.	3 "
25	41	Left chest one-third full of fluid.	14th "	Clear serum 1200 c.c.	"	Digitalis, squills, and strychnia.	6 "
26	27	Right chest half full of fluid.	14th "	Clear serum 800 c.c.	"	"	7 "
27	27	Right chest one-third full of fluid.	21st "	Clear serum 1020 c.c.	"	Strapping the chest.	5 "

No.	Age.	Condition of the chest on admission.	Day tapped.	Character of the fluid	Subsequent tappings.	Other treatment.	No. of days from first tapping to end of pleurisy.
28	25	Left chest half full of fluid.	21st day	Bloody ser. 850 c.c.	None.	None.	18 days; improved.
29	35	Right chest half full of fluid.	28th "	Clear serum 900 c.c.	"	Diuretic pill.	8 days.
30	24	Left chest full of fluid.	19th "	Clear serum 1200 c.c.	"	None.	11 "
31	24	Left chest half full of fluid.	7th "	Clear serum 1650 c.c.	"	Diuretic pill.	5 "
32	34	Right chest one-third full of fluid.	90th "	Turbid ser. 850 c.c.	"	Potassium iodide.	11 "
33	36	Right chest half full of fluid.	60th "	Turbid ser. 38 ounces.	"	Potassium iodide.	12 "
34	31	Right chest half full of fluid.	42d "	Clear serum 1400 c.c.	"	Potassium iodide.	10 "
35	13	Right chest two-thirds full of fluid.	21st "	Clear serum 800 c.c.	Clear serum 1300 c.c.	Potassium iodide.	29 "
36	51	Left chest three-fourths full of fluid.	28th "	Clear serum 1900 c.c.	Clear serum 1750 c.c.	Digitalin.	24 "
37	26	Right chest half full of fluid.	6th "	Clear serum 750 c.c.	None.	"	9 "
38	24	Right chest half full of fluid.	90th "	Clear serum 525 c.c.	"	Potassium iodide	9 "
39	24	Left chest two-thirds full of fluid.	14th "	Clear serum	"	Potassium iodide.	11 "
40	30	Right chest two-thirds full of fluid.	75th "	Clear serum 1675 c.c.	"	Potassium acetate and digitalis.	18 "
41	17	Left chest half full of fluid.	18th "	Reddish ser. 900 c.c.	"	Potassium acetate and digitalis.	12 "
42	24	Left chest half full of fluid.	21st "	Clear serum 300 c.c.	"	Potassium acetate and digitalis.	11 "
43	26	Left chest three-fourths full of fluid.	60th "	Reddish ser. 300 c.c.	"	Potassium acetate and digitalis.	28 "
44	34	Right chest three-fourths full of fluid.	14th "	Clear serum 1075 c.c.	"	Diuretic pill.	13 "
45	19	Left chest three-fourths full of fluid.	10th "	Clear serum 1575 c.c.	"	None.	9 "
46	20	Left chest full of fluid.	4th "	Reddish ser. 250 c.c.	"	Potassium iodide.	11 "
47	22	Left chest three-fourths full of fluid.	12th "	Clear serum 2000 c.c.	"	Potassium iodide.	10 "
48	18	Left chest half full of fluid.	13th "	Clear serum 200 c.c.	"	Potassium acetate and digitalis.	8 "
49	23	Left chest half full of fluid.	28th "	Turbid ser. 1400 c.c.	"	None.	15 "
50	50	Right chest half full of fluid.	8th "	Clear serum 575 c.c.	"	Potassium iodide.	4 "
51	57	Left chest half full of fluid.	28th "	Reddish ser. 1600 c c.	"	Potassium iodide and digitalis.	13 "
52	32	Right chest two-thirds full of fluid.	26th "	Clear serum 1325 c.c.	"	Potassium iodide and digitalis.	4 "
53	30	Right chest two-thirds full of fluid.	14th "	Reddish ser. 1100 c.c.	"	Potassium acetate and digitalis.	26 "
54	53	Left chest two-thirds full of fluid.	42d "	Clear serum 250 c.c.	Clear serum 250 c.c.	Digitalin.	38 days; improved.
55	29	Left chest half full of fluid.	14th "	Clear serum 1300 c.c	None.	None.	14 days.
56	45	Left chest full of fluid.	8th "	Clear serum 3700 c.c.	"	"	9 "
57	32	Left chest full of fluid.	14th "	Clear serum 375 c.c.	Clear serum 800 c.c.	"	39 "
58	22	Left chest half full of fluid.	20th "	Turbid ser. 700 c.c.	None.	Digitalin.	27 "
59	63	Right chest half full of fluid.	21st "	Clear serum 1000 c.c.	"	None.	16 "
60	30	Right chest full of fluid.	7th "	Clear serum 2000 c.c.	"	Potassium iodide and digitalis.	17 "

No.	Age.	Condition of the chest on admission.	Day tapped.	Character of the fluid.	Subsequent tappings.	Other treatment.	No. of days from first tapping to end of pleurisy.
61	40	Left chest three-fourths full of fluid.	90th day.	Clear serum 2200 c.c.	None.	Potassium iodide and digitalis	6 days.
62	8	Left chest half full of fluid.	3d "	Clear serum 675 c.c.	"	Potassium iodide and digitalis.	13 "
63	20	Left chest half full of fluid.	7th "	Clear serum 700 c.c.	"	None.	11 "
64	30	Right chest full of fluid.	7th "	Clear serum 1700 c.c.	600 c.c. 500 c.c.	"	16 "
65	24	Left chest half full of fluid.	21st "	Turbid ser. 250 c.c.	None.	"	9 "
66	22	Left chest half full of fluid.	16th "	Clear serum 1250 c.c.	1250 c.c. 750 c.c.	Potassium iodide and digitalis.	42 "
67	38	Right chest full of fluid.	42d "	Clear serum 900 c.c.	None.	Digitalin.	9 "
68	20	Left chest full of fluid.	14th "	Clear serum 1900 c.c.	800 c.c.	None.	9 "
69	33	Left chest three-fourths full of fluid.	7th mo.	Clear serum 1250 c.c.	None.	Digitalin.	12 "
70	50	Left chest full of fluid.	30th day.	Clear serum 1725 c.c.	Clear serum 2050 c c.	"	42 "
71	48	Left chest two-thirds full of fluid.	6th "	Clear serum 1100 c.c.	None.	"	16 "
72	26	Left chest three-fourths full of fluid.	14th "	Bloody ser. 800 c.c.	"	None.	14 "
73	39	Left chest full of fluid.	4th "	Clear serum 1300 c.c.	1500 c.c.	Digitalin.	10 "
74	19	Right chest two-thirds full of fluid.	12th "	Clear serum 1400 c.c.	None.	Diuretic pill.	15 "
75	47	Left chest half full of fluid.	15th "	Clear serum 375 c.c.	1200 c.c.	Potassium iodide and diuret. pill.	38 days; not improved.
76	29	Right chest full of fluid.	14th "	Clear serum 2100 c.c.	None.	Diuretic pill.	9 days.
77	29	Right chest half full of fluid.	1st "	Clear serum 270 c.c.	"	Diuretic pill.	8 "
78	[illegible]	Left chest half full of fluid.	10th "	Bloody ser. 2 ounces.	"	Diuretic pill.	6 "
79	36	Right chest half full of fluid.	21st "	Clear serum 880 c.c.	"	Diuretic pill	13 "
80	22	Left chest half full of fluid.	10th "	Clear serum 24 ounces.	"	None.	11 "
81	18	Left chest half full of fluid.	49th "	Clear serum 435 c.c.	"	Diuretic pill.	11 "
82	29	Left chest half full of fluid.	21st "	Clear serum 30 ounces.	"	Diuretic pill	17 "
83	42	Left chest full of fluid.	10th "	Bloody ser. 1800 c.c.	1950 c.c. 1500 c.c. 50 c.c.	None.	43 "
84	45	Right chest three-fourths full of fluid.	15th "	Purulent ser. 2200 c.c.	Turbid ser. 500 c.c.	"	12 "
85	27	Left chest half full of fluid.	11th "	Turbid ser. 950 c.c.	None.	"	5 "
86	30	Left chest half full of fluid.	22d "	Bloody ser 850 c.c.	"	"	8 "
87	48	Left chest half full of fluid.	18th "	Clear serum 320 c.c.	"	"	4 "
88	50	Right chest half full of fluid.	22d "	Clear serum 1600 c.c.	"	"	6 "
89	47	Right chest two-thirds full of fluid.	16th "	Clear serum 2750 c.c.	"	"	11 "
90	29	Left chest full of fluid.	14th "	Clear serum 2200 c c.	225 c.c.	"	15 "
91	17	Left chest half full of fluid.	22d "	Clear serum 1500 c.c.	None.	"	5 "
92	47	Right chest half full of fluid	9th "	Clear serum 1500 c.c.	"	"	7 "
93	38	Right chest three-fourths full of fluid.	37th "	Clear serum 1680 c.c.	"	"	10 "
94	48	Left chest half full of fluid.	30th "	Turbid ser 1600 c.c.	"	"	8 "
95	32	Left chest half full of fluid.	11th "	Clear serum 1100 c.c.	"	"	8 "
96	21	Left chest two-thirds full of fluid.	29th "	Clear serum 700 c.c.	"	"	6 "

No.	Age.	Condition of the chest on admission.	Day tapped.	Character of the fluid.	Subsequent tappings.	Other treatment.	No. of days from first tapping to end of pleurisy.
97	49	Left chest half full of fluid.	16th day.	Clear serum 850 c.c.	None.	None.	3 days.
98	28	Left chest two-thirds full of fluid.	16th "	Clear serum 1300 c.c.	"	"	4 "
99	25	Left chest three-fourths full of fluid.	19th "	Clear serum 2250 c.c.	"	"	3 "
100	29	Right chest full of fluid.	16th "	Clear serum 2700 c.c.	"	"	9 "
101	19	Left chest full of fluid.	60th "	Clear serum 400 c.c.	"	"	10 "
102	32	Left chest full of fluid.	21st "	Clear serum 800 c.c.	"	"	15 "
103	21	Right chest half full of fluid.	50th "	Clear serum 800 c.c.	"	"	6 "
104	24	Left chest half full of fluid.	11th "	Clear serum 1200 c.c.	Clear serum 1200 c.c.	"	12 "
105	40	Left chest half full of fluid.	60th "	Clear serum 1200 c.c.	Clear serum 1000 c.c.	"	12 "
106	55	Right chest half full of fluid.	19th "	Clear serum 1500 c.c.	Bloody ser. 100 c.c.	"	6 "
107	39	Right chest half full of fluid.	50th "	Clear serum 1400 c.c.	Clear serum 1000 c.c.	"	9 "
108	21	Left chest two-thirds full of fluid.	43d "	Clear serum 950 c.c.	Clear serum 950 c.c.	"	13 "
109	39	Left chest one third full of fluid.	90th "	Clear serum 500 c.c.	Clear serum 25 c.c.	"	6 "
110	48	Left chest one-third full of fluid.	90th "	Clear serum 800 c.c.	None.	"	4 "
111	27	Right chest two-thirds full of fluid.	28th "	Clear serum 900 c.c.	"	"	3 "
112	22	Left chest one third full of fluid.	46th "	Clear serum 600 c.c.	"	"	10 days; improved.
113	26	Left chest one-third full of fluid.	60th "	Bloody ser. 1000 c.c.	"	"	10 days.
114	23	Right chest half full of fluid.	13th "	Clear serum 900 c.c.	Clear serum 300 c.c.	"	9 "
115	16	Left chest full of fluid.	28th "	Clear serum 1100 c.c.	Clear serum 100 c.c.	"	6 "
116	37	Left chest half full of fluid.	11th "	Bloody ser. 100 c.c.	None.	"	Discharged; improved.
117	38	Left chest half full of fluid.	15th "	Clear serum 1300 c.c	Clear serum 750 c.c.	"	28 days.
118	24	Right chest half full of fluid.	30th "	Clear serum 250 c.c.	None.	"	7 "
119	18	Right chest half full of fluid.	6th "	Clear serum 900 c.c.	Clear serum 150 c.c.	"	9 days; improved.
120	23	Left chest one-third full of fluid.	10th "	Clear serum 1200 c.c.	None.	"	6 days.
121	3[illegible]	Left chest one-third full of fluid.	13th "	Clear serum 1300 c.c.	"	"	17 "
122	34	Left chest half full of fluid.	13th "	Clear serum 1500 c.c.	"	"	36 days; improved.
123	28	Left chest one-third full of fluid.	17th "	Bloody ser 100 c.c.	Clear serum 100 c.c.	"	22 days; improved.
124	25	Left chest two-thirds full of fluid.	17th "	Clear serum 1150 c.c.	1100 c.c. 1100 c.c.	"	35 days.
125	26	Right chest one-third full of fluid.	33d "	Bloody ser. 1000 c.c.	None.	"	5 "
126	29	Left chest one-third full of fluid.	34th "	Clear serum 1250 c.c.	"	"	6 "
127	7	Left chest one-third full of fluid.	17th "	Turbid ser. 300 c.c.	"	"	12 "
128	37	Left chest full of fluid.	9th "	Clear serum 1750 c.c.	Clear serum 1100 c.c.	"	9 "
129	35	Left chest one-third full of fluid	17th "	Clear serum 1300 c.c.	Clear serum 900 c.c.	"	12 "
130	58	Right chest two-thirds full of fluid.	20th "	Clear serum 1160 c.c.	None.	"	12 "
131	35	Left chest full of fluid.	31st "	Clear serum 1200 c.c.	Clear serum 1100 c.c. 250 c.c.	"	9 days; improved.
132	11	Right chest two-thirds full of fluid.	15th "	Cloudy ser. 400 c.c.	None.	"	21 days.
133	29	Right chest two-thirds full of fluid.	31st "	Clear serum 270 c.c.	"	"	8 "
134	29	Right chest full of fluid.	16th "	Clear serum 2100 c.c.	"	"	9 "

No.	Age.	Condition of the chest on admission.	Day tapped.	Character of the fluid.	Subsequent tappings.	Other treatment.	No. of days from first tapping to end of pleurisy.
135	19	Right chest half full of fluid.	19th day.	Bloody ser. 5 ounces.	None.	None.	4 days.
136	45	Right chest two-thirds full of fluid.	30th “	Greenish ser. 1560 c.c.	“	“	7 “
137	27	Left chest half full of fluid.	16th “	Brownish ser 315 c.c.	“	“	7 “
138	30	Right chest half full of fluid.	23d “	Clear serum 600 c.c.	“	“	8 “
139	47	Left chest half full of fluid.	31st “	Clear serum 900 c.c.	“	“	4 “
140	34	Right chest two-thirds full of fluid.	37th “	Greenish ser. 2640 c.c.	“	“	20 “
141	40	Right chest three-fourths full of fluid.	46th “	Clear serum 2160 c.c.	“	“	23 “
142	31	Left chest two-thirds full of fluid.	12th “	Clear serum 7 ounces.	“	“	6 days; improved.
143	50	Left chest half full of fluid.	29th “	Bloody ser. 200 c.c.	“	“	3 days.
144	33	Right chest two-thirds full of fluid.	72d “	Clear serum 900 c c.	“	“	7 “
145	48	Right chest two-thirds full of fluid.	17th “	Clear serum 300 c.c.	“	“	4 “
146	33	Left chest half full of fluid.	72d “	Clear serum 2000 c.c.	“	“	17 “
147	22	Left chest half full of fluid.	22d “	Clear serum 1250 c.c.	Clear serum 1100 c.c. 1000 c.c.	“	10 “
148	38	Right chest half full of fluid.	24th “	Brown ser. 400 c.c.	None.	“	6 “
149	23	Right chest three-fourths full of fluid	29th “	Clear serum 900 c.c.	“	“	6 “
150	48	Right chest half full of fluid.	20th “	Clear serum 1500 c.c.	“	“	22 “
151	28	Left chest two-thirds full of fluid.	61st “	Clear serum 1100 c.c.	Clear serum 750 c.c.	“	10 “
152	18	Left chest half full of fluid.	43d “	Clear serum 1000 c.c.	None.	“	4 “
153	31	Left chest half full of fluid.	11th “	Clear serum 1100 c.c.	450 c.c.	“	10 “
154	17	Right chest two-thirds full of fluid.	17th “	Clear serum 800 c.c.	None.	“	9 “
155	61	Right chest half full of fluid.	11th “	Clear serum 1600 c.c.	850 c.c.	“	13 “
156	24	Right chest two-thirds full of fluid.	15th “	Clear serum 600 c.c.	800 c.c.	“	30 “
157	46	Right chest half full of fluid.	20th “	Clear serum 1200 c.c.	480 c.c.	“	20 “
158	24	Right chest half full of fluid.	8th “	Clear serum 900 c.c.	None.	“	18 “
159	42	Left chest half full of fluid.	12th “	Clear serum 1250 c.c.	1350 c.c.	“	16 “
160	49	Left chest half full of fluid.	43d “	Turbid ser. 500 c.c.	125 c.c.	“	7 “
161	27	Right chest two-thirds full of fluid.	21st “	Clear serum 700 c.c.	20 c.c,	“	5 “
162	32	Left chest half full of fluid.	14th “	Clear serum 1000 c.c.	None.	“	1 “
163	38	Left chest two-thirds full of fluid.	17th “	Clear serum 1530 c.c.	64 c.c.	“	8 “
164	49	Left chest two-thirds full of fluid.	17th “	Clear serum 1920 c.c.	1856 c c.	“	6 “
165	29	Right chest two-thirds full of fluid.	54th “	Clear serum 1920 c.c.	480 c.c.	“	6 “
166	30	Right chest half full of fluid.	12th “	Bloody ser. 840 c.c.	None.	“	5 “
167	56	Left chest two-thirds full of fluid.	23d “	Clear serum 1728 c.c.	128 c.c.	“	7 “
168	18	Left chest two-thirds full of fluid.	14th “	Turbid ser. 1984 c.c.	None.	“	2 “
169	60	Right chest two-thirds full of fluid.	19th “	Clear serum 1920 c.c.	“	“	8 “
170	38	Right chest half full of fluid.	23d “	Turbid ser. 460 c.c.	“	“	17 days; improved.
171	22	Right chest two-thirds full of fluid.	13th “	Turbid ser. 1400 c.c.	“	“	2 days.
172	24	Left chest half full of fluid.	51st “	Turbid ser. 550 c c.	“	“	2 “

No.	Age.	Condition of the chest on admission.	Day tapped.	Character of the fluid.	Subsequent tappings.	Other treatment.	No. of days from first tapping to end of pleurisy.
173	8	Left chest full of fluid.	15th day.	Clear serum 600 c.c	None.	None.	1 day.
174	39	Left chest half full of fluid.	23d "	Clear serum 2200 c c.	"	"	2 days.
175	57	Left chest two-thirds full of fluid.	51st "	Clear serum 2100 c.c.	1600 c.c.	"	6 "
176	41	Left chest half full of fluid.	41st "	Clear serum 1475 c.c.	None.	"	1 day.
177	16	Left chest half full of fluid.	68th "	Turbid ser. 600 c.c	"	"	3 days.
178	30	Left chest two-thirds full of fluid.	24th "	Clear serum 1100 c.c.	"	"	2 "
179	30	Right chest one-third full of fluid.	14th "	Bloody ser. 380 c.c.	"	"	2 "
180	29	Left chest half full of fluid.	6th "	Clear serum 200 c.c.	"	"	1 day.
181	23	Right chest half full of fluid.	31st "	Bloody ser. 875 c.c.	"	"	1 "
182	26	Whole right chest full of fluid.	32d "	Turbid ser. 900 c c.	1800 c.c. 576 c.c.	"	23 days.
183	12	Right chest two-thirds full of fluid.	11th "	Clear serum 1000 c.c.	None.	"	13 "
184	25	Left chest half full of fluid	7th "	Clear serum 1000 c.c.	"	"	10 "
185	25	Right chest half full of fluid.	22d "	Clear serum 900 c.c.	100 c.c.	"	8 "
186	37	Left chest three-fourths full of fluid.	8th "	Clear serum 2100 c.c.	500 c,c. 100 c.c.	"	39 "
187	38	Left chest full of fluid.	56th "	Clear serum 1100 c.c.	2000 c.c. 1350 c.c. 400 c.c.	"	34 days; improved.
188	38	Right chest two-thirds full of fluid	56th "	Clear serum 1100 c c.	500 c.c.	"	15 days.
189	50	Left chest two-thirds full of fluid.	23d "	Clear serum 1100 c.c.	Clear serum 960 c.c.	"	10 "
190	23	Right chest two-thirds full of fluid.	17th "	Clear serum 1350 c.c.	None	"	15 "
191	57	Left chest two-thirds full of fluid.	21st "	Clear serum 1100 c.c.	3400 c.c. 1450 c.c. 525 c.c.	"	36 days; improved.
192	61	Left chest half full of fluid.	8th "	Clear serum 1600 c c.	None.	"	7 days
193	26	Left chest half full of fluid.	9th "	Clear serum 1900 c.c.	400 c.c.	"	8 "
194	22	Left chest two-thirds full of fluid.	7th "	Clear serum 1600 c c.	150 c.c.	"	11 "
195	35	Right chest half full of fluid.	29th "	Clear serum 2500 c c.	None.	"	32 "
196	71	Right chest half full of fluid.	5th "	Clear serum 1150 c.c.	"	"	14 "
197	37	Left chest half full of fluid.	23d "	Clear serum 550 c.c.	"	"	25 days; improved.
198	26	Right chest half full of fluid.	17th "	Clear serum 300 c.c.	400 c.c.	"	23 days.
199	40	Left chest two-thirds full of fluid.	24th "	Clear serum 300 c.c.	500 c.c. 550 c.c.	"	24 days. improved.
200	39	Left chest two-thirds full of fluid.	9th "	Clear serum 1150 c.c.	1650 c.c. 550 c.c.	"	28 days. improved.

PLEURISY WITH THE PRODUCTION OF SERUM, FIBRIN, AND PUS. EMPYEMA.

Lesions.—The inflammation regularly involves the whole of the pleura on one side of the chest, less frequently a circumscribed portion of the pleura. The inflammation follows two different forms:

1. The pleura is coated with fibrin and pus, and its cavity contains purulent serum, but the pleura itself is but little changed.

This form is most common in children.

2. The pleura is coated with fibrin and pus, its cavity contains purulent serum, and in addition the pleura itself is much changed. It is split up by great numbers of new cells, so that it resembles granulation tissue.

In either case the fluid accumulates in the lower part of the pleural cavity, pushing the lung upward and toward the vertebral column; or in the posterior part of the pleural cavity, pushing the lung forward; or it is sacculated in any part of the pleural cavity.

The lung is usually much compressed. In old cases the pleura becomes much thickened, and may be infiltrated with the salts of lime.

The suppurative process may extend from the pulmonary pleura to the lung, and the pus then escape at intervals from the bronchi; or it may extend from the costal pleura to the wall of the chest and the pus escape externally.

In a few cases the inflammatory products and the superficial layers of the pleura become gangrenous.

The micro-organisms regularly found are either streptococci or pneumococci. There seems to be no special difference in the clinical symptoms whether the infection is effected by one or the other of these organisms.

Symptoms.—The inflammation may be primary, after exposure to cold, or without discoverable cause. The patients are suddenly attacked with chills, a high temperature, marked prostration, headache, pains in the back and limbs, pain over the inflamed pleura, shallow and painful breathing, sometimes cough.

The symptoms may continue acutely and the patients die in a short time, or they may subside and the inflammation pass into the chronic condition.

It is difficult to distinguish this form of empyema from lobar pneumonia. It is apt to occur at a time when pneumonia is prevailing; the constitutional symptoms are the same; the patients are too sick to admit of a thorough examination of the chest, and the physical signs are often misleading. When the empyema is sacculated the physical signs are especially obscure.

CASE X. *Acute Empyema; Death.*—Female, 35 years old, January 3, 1879. She was well until one week ago; then she had pain in the right side, cough, chill and fever. To-day her temperature is 104.5° F., her pulse 120, her breathing 36.

On the right side there are bronchial breathing and coarse râles over the upper part of the chest, flatness and absence of voice and breathing over the lower part.

Seventeenth day she died.

Autopsy.—The right pleural cavity was two-thirds full of turbid serum; the pleura was coated with pus and fibrin; the bronchi of both lungs were congested and full of pus.

CASE XI. *Acute Empyema; Pericarditis; Sudden Death.*—Male, 37 years old, January 27, 1882. Eight days ago the man was attacked with chill, cough and yellow expectoration, pain low down in the right side, fever, sleeplessness, and dyspnœa.

He is a large, strong man, the face flushed, the tongue coated; temperature 99.3° F., pulse 124, breathing 52.

Tenth day: Sixteen ounces of thin pus were aspirated from the seventh space, just in front of the right axillary line.

Fifteenth day: Pulse 120, breathing 44, temperature 103° F. On the right side in front there is flatness from the second rib down. Behind there is flatness from the top of the lung to the angle of the scapula.

Twenty-seventh day: Pulse 108, breathing 48, temperature 101° F. The man sits up, eats solid food, but sleeps badly.

Thirty-first day: The man sat up all day and at 10 o'clock in the evening suddenly died.

Autopsy.—The visceral and parietal pericardium were thickened and coated with fibrin. The valves of the heart were normal.

There was a sacculated collection of pus occupying the upper part of the right pleural cavity, extending from the second to the fifth rib. Both lungs were œdematous; the other viscera were normal.

There are cases in which the empyema, although extensive, is only one of the evidences of a general streptococcus or pneumococcus poisoning, and yet no original focus for the poisoning can be found.

CASE XII.—A girl, 3 years old, is said to have been well until the morning of September 16. Then she had a convulsion, and in the afternoon a second convulsion. After the second convulsion she was semi-comatose; temperature 99° F., pulse 192, breathing 48; urine 1032, no albumin. In the evening she had two more convulsions, one of them lasting for twenty minutes.

On the second day she became conscious, temperature 99° F., pulse 168, breathing 36.

On the third day she passed one round worm after taking a vermifuge; temperature 97.5° to 97.6° F., pulse 156 to 144, breathing 36 to 48; a little coughing.

Fourth day: The temperature rose to 102° F., and afterward ranged between 95° and 101° F.; the pulse and breathing continued to be rapid. There were no more cerebral symptoms.

On the seventh day there were found the physical signs of fluid in the right chest.

On the eleventh day the child died.

Autopsy.—Both the brain and the pia mater are congested, with some increase of serum. The pericardium is coated with fibrin and contains purulent serum. Both pleural cavities are about half full of purulent serum, the pleuræ are coated with fibrin, the lungs are compressed but not inflamed. All the other viscera normal, nor could any lesions be found in any other part of the body.

The empyema may be secondary to a pleurisy with effusion. The character of the inflammation changes slowly or rapidly as the pus cells are added to the fibrin and serum.

The temperatures run higher and the patients lose flesh and strength rapidly.

As early aspirations with clean instruments have become more common this change in the character of the inflammation is getting to be less frequent.

CASE XIII. *Empyema following Pleurisy with Effusion.*—Male, 30 years old, March 11, 1881. Six and one-half weeks ago he was exposed to cold and wet, and had a cold in the head. Nine days after this he had a single chill, followed by pain in the left chest, and fever with delirium. These acute symptoms only lasted a few days, but he did not afterward regain his strength. Five weeks ago the chest was aspirated and clear serum drawn off. After this he was better for a few days, but then became much worse, with loss of appetite and strength, and profuse sweating.

Now the man is emaciated; over the whole left chest behind there is flatness on percussion; bronchial voice and breathing from the spine of the scapula to its angle; below this the voice and breathing are absent. In front there is dulness from the clavicle to the third rib. The apex of the heart is an inch and a half to the right side of the right nipple. Pulse 150, breathing 40, temperature 103° F.

March 12: Aspirated thirteen ounces of thin pus. After this the man did well, with normal temperatures, until March 19, when it was found that the chest had filled up with fluid. After this the man had nearly normal temperatures, was doing well, and out of bed until April 3. Then the temperature rose to 103° F., and an abscess was forming beneath the skin, just below the angle of the scapula.

After this the man grew rapidly worse until April 6. Then an opening was made in the chest, in the eighth interspace, a little to the left of the scapula. A second opening was made in the seventh interspace, an inch in front of the axillary line; about ninety ounces of pus escaped. A large drainage tube was passed through the chest, emerging from each opening.

April 15: The man sits up to-day for the first time.

May 22: The tube has been removed and both openings in the chest have closed.

June 10: The man is discharged well.

After lobar pneumonia it is not uncommon to have a purulent pleurisy, and in New York this disposition is decidedly more marked during some winters than it is in others. The pleurisy is regularly developed as the pneumonia is subsiding, or a few days after defervescence. As the pleurisy is developed the temperature goes up again and the patients begin to run down. The physical signs are the regular ones of fluid in the pleural cavity, or they are irregular. The irregular physical signs are: either that we hear bronchial voice and breathing and subcrepitant râles below the level of the fluid, or that with sacculated collections of purulent fluid the signs of fluid in the lower part of the pleural cavity are absent.

In the broncho-pneumonia of children a very small collection of pus at the root of the lung will keep a child sick for weeks and months.

CASE XIV. *Empyema after Lobar Pneumonia; Improvement and Sudden Death.*—Male, 26 years old, January 8, 1886. A healthy, temperate young man.

Two days ago he was suddenly attacked with dyspnœa, pain in the right side, and great prostration.

Now the man is well nourished, face flushed, tongue coated, pain over lower part of the right chest, and great dyspnœa. Pulse 114, breathing 36, temperature 102° F.

Over the lower part of the right chest, behind, dulness, subcrepitant râles, bronchial voice and breathing.

Fifth day: Breathing 36, temperature 105° F., dulness and crepitant râles over the right middle lobe.

Eighth day: Temperature 101° F., delirious. Over the whole of the right lung, behind, flatness, absence of voice and breathing.

Fourteenth day: Aspirated twenty-seven ounces of purulent serum from the right side of the chest.

Seventeenth day: Aspirated eleven ounces of purulent serum.

Twentieth day: Aspirated twenty-one ounces of purulent serum.

Twenty-seventh day: Aspirated thirty-eight ounces of purulent serum.

Thirty-eighth day: Aspirated fifty-six ounces of purulent serum.

During all this time the man was septic, with high temperatures, sweating, rapid and feeble pulse, dry tongue, delirium, and rapid emaciation.

Thirty-ninth day: An opening was made in the eighth interspace, in the line of the angle of the scapula, and a drainage tube was put in.

After this the man improved rapidly, gained flesh and strength, and was up and out of doors, but the wound was not closed.

One hundred and fourteenth day: While the house physician was dressing the wound, and the patient was talking cheerfully and saying he had gained ten pounds in the last week, he suddenly fell forward and was dead.

Autopsy.—The brain and its membranes were normal. The left lung was a little congested; the right lung was completely adherent to the chest wall and diaphragm, except along the line of the drainage tube. The upper and middle lobes were well aërated. The lower lobe was dense and imperfectly aërated.

There was no cavity left in the pleura, except a narrow tract around the drainage tube.

The heart: both right and left ventricles were dilated, with thin walls. The valves were normal. All the cavities were distended, with red and yellow clots.

The liver was large and congested.

The spleen was large and congested.

The stomach and intestines were normal.

The kidneys were in a condition of chronic congestion.

There is a form of empyema which is rare and not well understood. There seems to be an additional infection to the one causing the purulent inflammation, which produces gangrene of the inflammatory products. The patients give the most marked evidence of septic poisoning.

CASE XV. *Gangrenous Empyema.*—Female, 22 years old, October 27, 1897. Three days ago she began to have pain in the left side of the chest, but continued to work until yesterday, when the pain was so severe that she went to bed. There was no cough and no chill.

Now patient is well nourished, with moderate prostration and moderate dyspnœa.

Over the left lung, behind, from midscapula to base, there is dulness on percussion and diminished breathing.

Eighteenth day: She looked septic; the expectoration has a gangrenous color; there is flatness over the lower part of the left lung.

Twentieth day: Aspirated 800 c.c. of very foul pus.

Twenty-second day: Resected six inches of the eighth rib on the left side and put in a drainage tube. After this she steadily improved.

Fiftieth day: The drainage tube was removed.

Sixtieth day: She was discharged cured.

Abscesses in the wall of the thorax, in the liver, in the abdominal cavity, or in the lung may rupture into the pleural cavity and set up a purulent inflammation.

The physical signs of empyema are the same as those of pleurisy with effusion, but sacculation of the fluid and irregular physical signs are more common.

The Course of the Disease.—Of the acute primary cases a considerable number go on rapidly and terminate fatally within a short time, but besides these there are others in which the first acute symptoms subside and the disease pursues a chronic course.

Of the secondary cases the larger number pursue a subacute course, with afternoon fever, dyspnœa, cough, and gradual loss of flesh and strength. In some the lung is perforated and the pus coughed up from time to time through the bronchi; in some the wall of the chest is perforated and the pus imperfectly evacuated.

Recovery with absorption or with perforation is very rare. But in some patients there is a partial recovery, most of the pus is absorbed, but there is set up an interstitial pneumonia, with more or less bronchitis, which goes on indefinitely.

On the other hand, recovery after a proper surgical operation is almost the rule.

Treatment.—Twenty-five years ago the treatment of empyema was very unsatisfactory and the mortality of the disease large. Some of the patients were allowed to go on under medical treatment for months and years. Then we saw the cases with great thickenings of the pleura, with calcific plates in the pleura, with spontaneous openings through the chest wall, with secondary tubercular inflammations of the lung, and with waxy degeneration of the liver, spleen, and kidneys. The attempts at surgical interference were also unsatisfactory, because it was not recognized that the patients were usually reinfected after the operation.

At the present time the rules for treatment are definitely determined and give satisfactory results.

In children under ten years of age a certain number of cases can be cured by aspiration alone. If, however, the inflammation continues after three aspirations, the chest must be opened and drained. It is not usually necessary to remove any of the ribs.

In sacculated purulent pleurisy, if it is of small size, aspiration alone will often effect a cure. It is curious to see the complete cessation of serious symptoms after the aspiration of only half an ounce of pus.

In adults with an empyema of any size the chest must be opened and drained and one rib resected. Different surgeons have different methods of operating, but they all get good results, if the pleura is not reinfected.

As I have watched these patients for years after operation, it has seemed to me that the most symmetrical and strongest chests are obtained by taking out the whole length of the first rib below the angle of the scapula.

After a successful operation the purulent discharge should nearly cease by the end of a week, and the drainage tube be removed at the end of four weeks.

The principal danger after operation is that of reinfection of the pleura.

If there is any purulent discharge from the pleura at the end of four weeks the drainage tube should not be allowed to remain, but another rib should be resected.

The cases of empyema I have already given show the poor results of the earlier inefficient treatment. The following cases are fair examples of routine treatment at the present time:

CASE XVI. *Empyema after Pneumonia.*—Male, 21 years old, tailor, October 15, 1897.

Three weeks ago he was sick with pneumonia of the left lung for seven days. Following this the cough and pain continued, with irregular chilly feelings, sweating, and loss of strength.

Now the man is pale, emaciated, temperature 102.6° F., pulse 96, breathing 28. Over the upper lobe of the left lung, in front and behind, there is dulness on percussion, with exaggerated voice and breathing. Over the lower part of the left lung there is flatness on percussion, with absence of voice, breathing, and vocal fremitus.

October 19: Two and a half inches of the left eighth rib below the scapula were resected and two drainage tubes inserted. (See Fig. 21.)

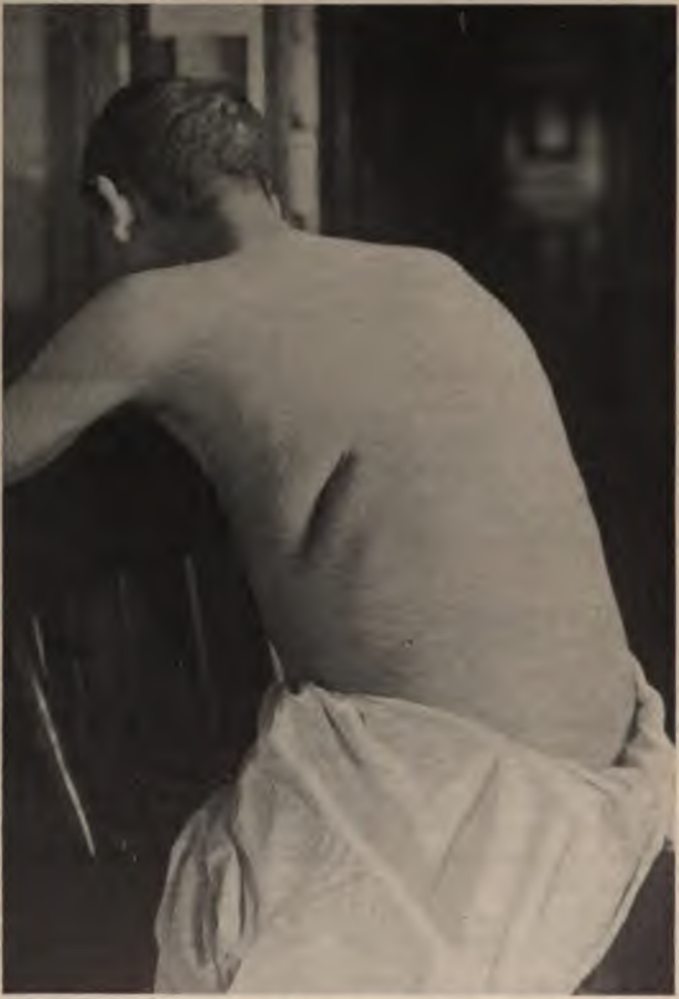
Fourth day after operation: Dressing changed for first time.

Eleventh day after operation: Dressing changed second time; the drainage tubes were shortened.

Nineteenth day after operation: The dressing changed the third time and one tube removed.

Twenty-seventh day after operation: The dressings changed the fourth time and the other tube removed.

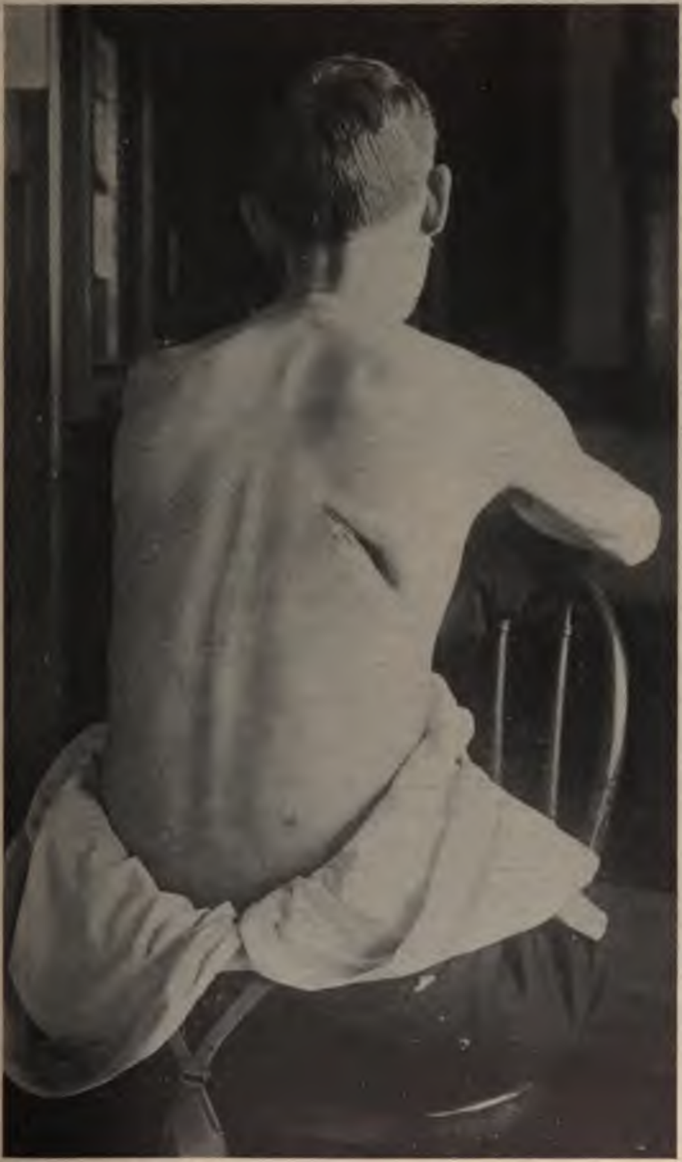
FIGURE 21.



Empyema after pneumonia. Appearance of healed wound.

Fortieth day after operation: Discharged cured. (See Fig. 21.)

FIGURE 22.



Empyema. Appearance of healed wound.

CASE XVII. *Empyema.*—Male, 48 years old, Oct. 14, 1897. One month before admission he had sharp pain in the right side, lasting for one day. Since then the cough has been troublesome, he has had irregular chilly feelings, and loss of flesh and strength.

Two weeks ago a needle in the right chest brought out semipurulent fluid.

October 14: The man is well nourished; no dyspnœa; temperature 101° F., pulse 88, breathing 24. Over the right chest, behind, from one inch below the spine of scapula down, absence of voice and breathing and of vocal fremitus.

October 16: Aspirated 500 c.c. thick pus.

October 17: Resected four inches of the right seventh rib. (See Fig. 22.)

Sixth day after operation: Dressing changed for the first time and the cavity washed out.

Tenth day after operation: Dressing changed for the second time and cavity washed out.

Eighteenth day after operation: Dressing changed for the third time.

Twenty-fourth day after operation: Dressing changed for the fourth time; one tube removed.

Twenty-ninth day after operation: Dressing changed for the fifth time; a second tube removed.

Forty-fourth day after operation: Discharged cured. (See Fig. 22.)

CASE XVIII. *Sacculated Empyema.*—A girl, 4 years old, October 29, 1897.

Five weeks ago she began to cough and complain of pain in the left side. She was chilly, feverish, and short of breath.

October 29: Temperature 103° F., breathing 44, pulse 160. On the left side, between the scapula and the vertebral column, flatness and absence of voice and breathing. Below the scapula, pulmonary resonance and absence of breathing. (See Fig. 23.)

November 3: Aspirated 250 c.c. of thick pus in the fifth interspace, between the scapula and the vertebræ.

November 11: Resected one inch of the fifth rib between the scapula and the vertebræ.

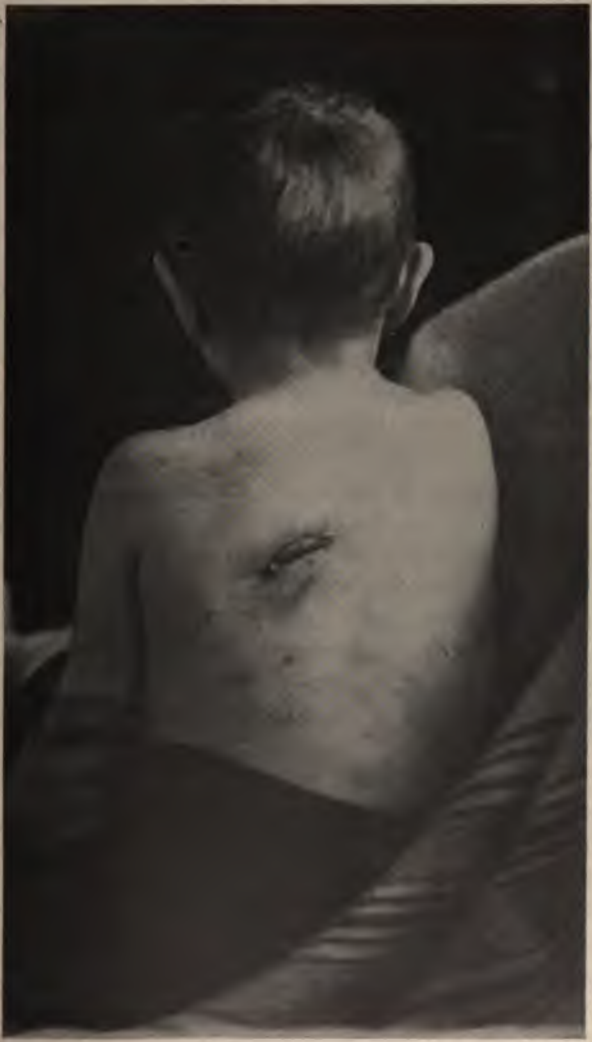
Seventeenth day after operation: The tube was removed.

Thirty-second day after operation: She was discharged cured. (See Fig. 23.)

CHRONIC PLEURISY WITH ADHESIONS.

This is a form of pleurisy which is not usually described. I do not mean by it the old adhesions which are found at so many autopsies, but a chronic productive inflammation of the pleura.

FIGURE 23.



Sacculated empyema. Appearance of healed wound.

It is an inflammation chronic from the outset, not producing

The lungs: The bronchi are congested and coated with muco-pus. In the right lung there are small areas of red hepatization.

The left lung is compressed, the lower two-thirds of the pleural cavity is filled with reddish serum. The pulmonary, costal, and mediastinal pleuræ are thickened, studded with white miliary tubercles, and coated with a thick layer of fibrin. Vertical sections of the costal pleura show the tubercles, for the most part, in the outer layers, next the ribs. The whole pleura is thickened, with many small polygonal cells in rows.

The liver weighs five and one-half pounds; small nodular cirrhosis.

The spleen is large and soft.

The kidneys are normal.

The stomach and small intestines are pale and covered with mucus.

THE PRIMARY NEW GROWTHS OF THE PLEURA.

The bibliography of endothelioma of the pleura is given by Glockner, *Zeitschrift der Heilkunde*, xvii., p. 209.

The number of cases of primary tumors of the pleura so far reported is small, so that I think it is worth while to report four cases which have been under my own observation.

It is generally agreed that these tumors are endothelial growths which originate in the lymphatics of the pleura. There is a new growth of cells from the endothelium of the lymphatics and an increase in the size and number of the lymphatics. At the same time the pleura itself is split up by an extensive new growth of cells, and there is a large transudation of blood serum rich in fibrino-plastic substances. In the earlier stages of the growth there are no tumors, only a diffuse thickening of the pleura, and, embedded in this, tubules lined or filled with endothelial cells. Later tumors are formed of different sizes, projecting inward, composed of a basement substance containing tubules or irregular cavities filled with polygonal cells. The pleura is coated with fibrin, and large quantities of serum collect in the pleural cavity. The inflammatory changes in the pleura are so marked that they may easily mask the new growth. In its earlier stages the real character of the lesion may be overlooked.

The new growth and the inflammatory changes may be confined to one pleura, they may involve both pleuræ, or the peritoneum also may be affected in the same way.

The history is that of a pleurisy with effusion, which is unusually severe and which terminates fatally.

During the first weeks it is hardly possible to distinguish these cases from ordinary cases of pleurisy with effusion. The story of the invasion, the behavior of the patients, the physical signs are all the same. Only the bloody serum and the low temperatures are suspicious.

But as the weeks follow each other, the loss of flesh and strength and the appearance of the patient make one think more and more of malignant disease.

CASE XXI. *Primary Endothelioma of the Pleura.*—Female, 53 years old, September 15, 1880. Four weeks ago she caught cold. Since that time she has had a cough with scanty expectoration; has been chilly and feverish, with dyspnœa, pain and oppression on the right side, and loss of flesh and strength.

She is now feeble and emaciated, with a little cough, dyspnœa, and pain and oppression over the right chest; pulse 100, breathing 24, temperature 98° F.; urine 1024, no albumin or casts. The physical signs are those of fluid in the left pleural cavity.

September 18: Fifty-five ounces of clear serum were aspirated from the left pleural cavity; temperature 99° F.

October 12: The woman feels better and is out of bed.

November 19: A quantity of dark-brown, slightly purulent fluid was aspirated from the left chest.

December 14: The chest was again aspirated and the same dark-brown fluid drawn off.

December 21: She died rather suddenly.

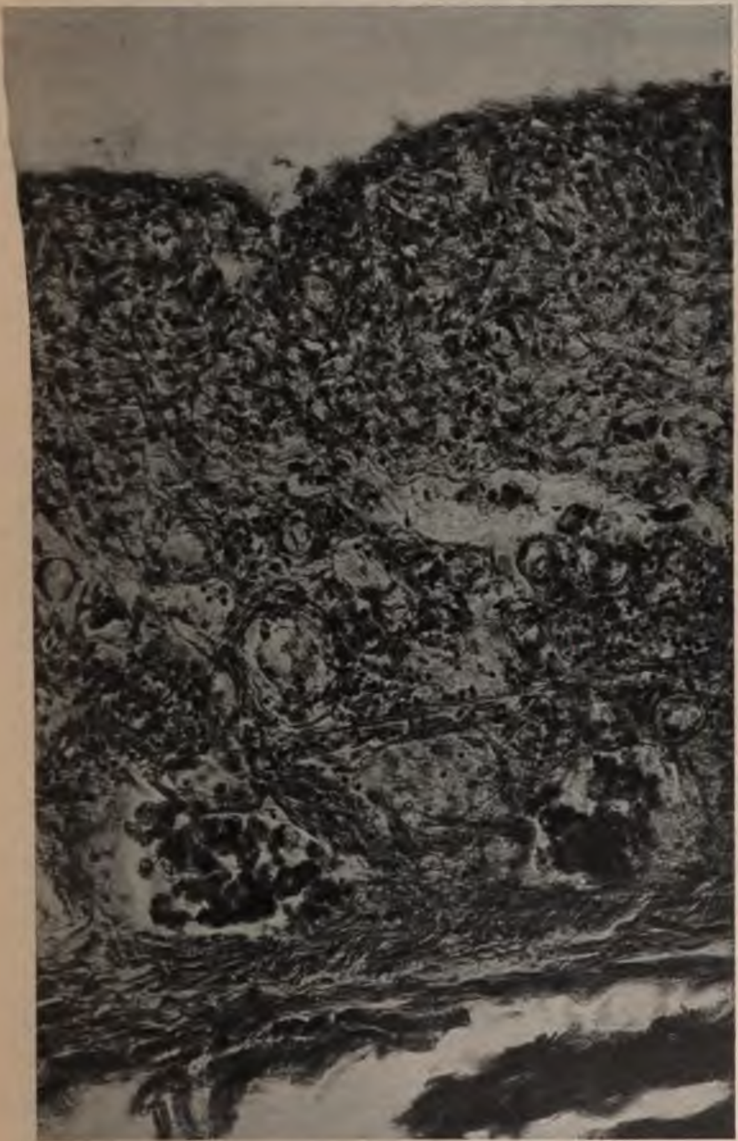
Autopsy.—The brain was not examined. The heart was normal; it was displaced to the right of the sternum.

The right lung was normal.

The left pleural cavity was nearly full of bloody, purulent serum. The lung was compressed against the vertebral column. The pleura was thickly coated with fibrin.

Vertical sections of the costal pleura show on the inner surface a layer of fibrin and pus. Beneath this is a layer of reticulated connective tissue. In this are tubules filled with large, polygonal nucleated cells. (See Fig. 25.) Beneath this layer is the pleura itself, its cells increased in size and number. In flat sections the tubules look like lymphatics.

FIGURE 25.

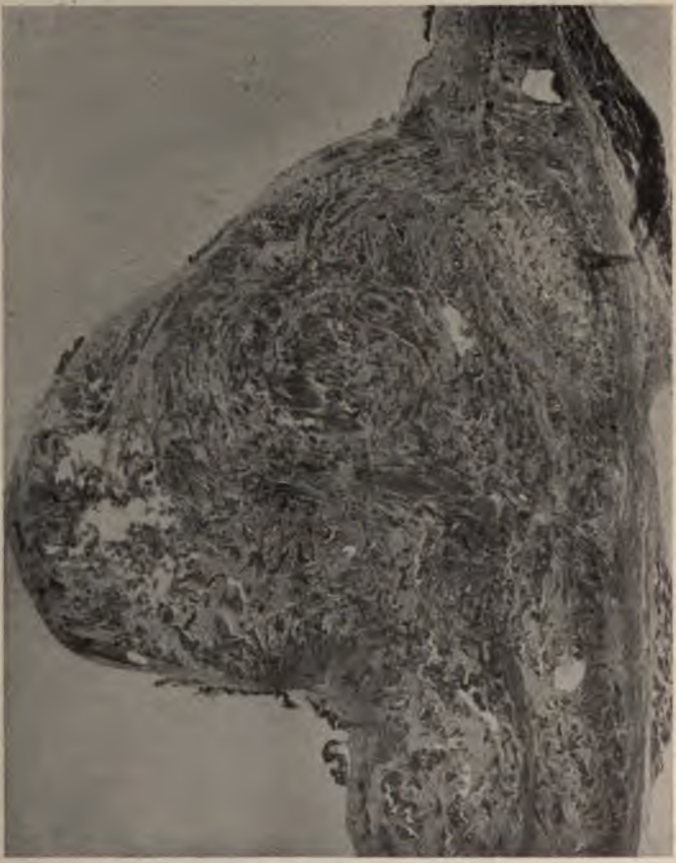


Primary endothelioma of the pleura. Vertical section showing the thickening of the pleura and the tubules of the new growth.

The liver and spleen are normal.

The kidneys are of medium size, their capsules adherent, their surfaces finely nodular.

FIGURE 26.



Primary endothelioma of the pleura. Vertical section of a small nodule of the new growth.

CASE XXII. *Primary Endothelioma of the Pleura.*—Male, 63 years old, June 12, 1883.

Two weeks ago he caught cold and had cough; pain in the right side of the chest.

He came into the hospital with these symptoms and with a friction sound over the lower lobe of the right lung.

He remained in the hospital until June 16 and was then discharged improved.

July 25 he was readmitted. The pain in the right side has continued and he has had to stop work. There is flatness on percussion, with absence of voice and breathing on the right side of the chest from the angle of the scapula down.

September 15: A little bloody serum was aspirated from the right chest.

October 3: Thirty-seven ounces of bloody serum were aspirated from the right chest.

After this the man steadily lost flesh and strength until October 8, when he died.

Autopsy.—The brain was not examined.

The heart was normal.

The left lung was emphysematous.

The right lung was confined by old adhesions to the upper third of the thorax; the lower lobe was compressed.

The lower part of the pleural cavity was full of bloody serum. The costal, pulmonary, and diaphragmatic pleuræ are coated with fibrin, thickened and studded with small and large white nodules. (See Fig. 26.) The nodules are composed of a connective-tissue stroma enclosing irregular cavities filled with flat, nucleated cells.

The liver is fatty.

The stomach, intestines, and spleen are normal.

The kidneys show the changes of chronic nephritis with commencing atrophy.

CASE XXIII. *Primary Endothelioma of the Pleura.*—Male, 60 years old, November 27, 1884.

The man says that he was at work until six weeks ago. Then he began to have pain on both sides of the chest; dyspnœa on exertion; cough with muco-purulent and bloody sputum, and occasionally vomiting.

Now he is feeble, emaciated, stupid; pulse 94, breathing 18, temperature 98° F. There is flatness and absence of breathing over the whole right lung and behind. The man lay quietly in bed, growing more feeble and emaciated every day, until December 11, when he died.

Autopsy.—The left pleural cavity contains a little serum. The left lung is coated with old adhesions, and there are small white, flat nodules in the pulmonary pleura.

The right pleural cavity is distended with bloody serum. The lung is compressed against the vertebral column. The costal and diaphragmatic pleuræ are thickly studded with large and small white nodules.

The growth in the pleura is composed of polygonal cells, arranged in tubules.

The pleura is split up and filled with connective-tissue cells. There are very marked changes in the endothelium of the costal pleura. The cells look like cylindrical cells and are in masses.

The heart is small; the aortic and mitral valves are calcified.

The liver: There are old adhesions over its entire surface. It contains several large white nodules, some of them continuous with nodules in the diaphragm.

The spleen is large and firm; it contains a large white nodule.

The peritoneal cavity contains bloody serum. There are nodules of new growth in the omentum and the mesentery.

The stomach is small and congested.

The intestines are normal.

The kidneys are normal.

CASE XXIV. *Primary Endothelioma of the Pleura.*—Male, 45 years old, June 6, 1884.

Three weeks ago he began to lose appetite and feel weak. One week later he had dyspnœa on exertion, slight cough with mucous expectoration, and pain in the chest.

June 6: There is flatness on percussion and absence of voice and breathing over nearly the whole of the right chest.

June 15: Nineteen ounces of bloody serum were drawn off from the right pleural cavity. His temperature has ranged from 98° to 100° F.

June 17, he died suddenly.

Autopsy.—The brain was not examined.

The heart was normal.

The right lung was compressed against the vertebral column. The pleural cavity was distended with bloody serum. The costal and diaphragmatic pleuræ are irregularly thickened and there are small white nodules in the costal and pulmonary pleuræ.

The left pleural cavity is half full of bloody serum. The costal pleura is thickened and congested; there are small white nodules in the pulmonary pleura.

The costal pleura of the right side is thickened and converted into granulation tissue with numerous blood vessels. In the deep layers of the pleura are small cavities filled with nucleated polygonal cells; in other places the cavities are larger and occupy most of the thickness of the pleura.

The liver, spleen, and kidneys are normal.

The peritoneal cavity contains turbid serum and the peritoneum is thickly studded with small white nodules.

The intestines are normal.

BRONCHITIS.

Causes.—Inflammation of the bronchi occurs at all ages. The acute form is more common in children; the chronic form is more common with emphysema, with heart disease, and in old persons. Persons living in cities, those who are much confined to the house, and those whose health has been enfeebled are especially liable to the disease.

There is in some persons a very well-marked predisposition to inflammation of the bronchi. This is especially marked in children and in young persons, in whom there seems to be a peculiar vulnerability of the mucous membrane of the bronchi, so that year after year they suffer from attacks of acute bronchitis. As they grow older this condition may disappear.

As exciting causes of bronchitis we recognize exposure to cold and wet, the inhalation of irritating gases and dusts, and of the vegetable substances and odors which cause hay fever.

Measles, whooping cough, and many of the infectious diseases are often complicated by bronchitis.

At the present time a large number of the cases of bronchitis seem to be caused by the poison of influenza.

1. The Acute Catarrhal Bronchitis of Adults.

Definition.—An acute exudative inflammation of the mucous membrane of the bronchi.

Lesions.—The inflammation involves the trachea, the larger bronchi, and the medium-sized bronchi, not as often the smaller ones. As a rule the bronchi of both lungs are equally inflamed. The mucous membrane of the bronchi is congested and swollen; at first it is dry, afterward coated by an increased production of mucus. There are also desquamation of the epithelial cells, emigration of white blood cells, diapedesis of red blood cells. The lumen of the bronchi may be narrowed either by the congestion and swelling of the mucous membrane or by the contraction of the muscular coat.

Symptoms.—The mild cases of acute bronchitis are attended with cough accompanied by scanty mucous sputa, by pain over the sternum and a moderate feeling of indisposition. There may be no physical signs, or a few coarse râles, or sibilant and sonorous breathing. The natural duration of the inflammation seems to be about a week, but it is often prolonged for a much longer time.

In the more severe cases there is a troublesome cough with mucous or muco-purulent sputum, often streaked with blood. In some cases the invasion is acute, with fever and sometimes chills; in others it is gradual, the cough preceding the fever by several days. The febrile movement is regularly lower in the morning and higher in the afternoon, running between 100° and 102°F. The patients complain of more or less pain about the sternum and the front of the chest. This pain seems to be due partly to the bronchitis and partly to the muscular effort of coughing. In the bad cases there may be severe dyspnœa.

The physical signs are: harsh breathing, sibilant and sonorous breathing, and coarse and subcrepitant râles. The râles are either diffuse over both lungs or localized.

1. A mild bronchitis lasts for about two weeks, the cough continuing longer than the fever.

Case I. *Acute Bronchitis, Mild.*—Male, 20 years old, February 22, 1896.

Ten days before his admission he was attacked with chill, general muscular soreness, cough with profuse mucous expectoration, and slight dyspnœa.

Now, temperature 100° F., pulse 60, breathing 20. There is harsh breathing, with subcrepitant râles over both lower lobes.

The man was put to bed, on a milk diet, with one-quarter grain of ipecac every hour for six doses. This medication was repeated for two days.

Fourteenth day: He was discharged cured.

CASE II. *Acute Bronchitis, Mild.*—Female, 15 years old, March 18, 1896.

On the day of her admission she began to have chilly feelings and muscular pains, with coryza and headache. Temperature 100° F., pulse 114, breathing 24. Over both lungs sonorous breathing and subcrepitant râles.

She was put to bed, on a milk diet.

Fourth day: She was discharged cured.

This ordinary picture of an acute bronchitis is often modified in a variety of ways.

2. There are cases with higher temperatures, more marked prostration, and of longer duration. Many of these cases have the bronchitis as the lesion of epidemic influenza, and the temperature and prostration belong partly to the influenza.

CASE III. *Acute Bronchitis, More Severe.*—Male, 41 years old, April 16, 1896.

On the day before admission he had a chill, followed by cough with scanty, mucous expectoration and pain in the right side.

Now, temperature 103° F., pulse 90, breathing 28. Over the right lower lobe subcrepitant râles.

Third day: Temperature 102° F.

Fourth day: Temperature 102° to 100° F.

Fifth day: Temperature 100° F.

Sixth day: Temperature normal.

The man was put to bed, on a milk diet, and had no other treatment.

Seventh day: He was discharged cured.

CASE IV. *Acute Bronchitis, More Severe.*—Female, 18 years old, May 4, 1896.

Four days ago she began to have headache, nausea and vomiting, and cough with mucous expectoration. Now she feels quite ill; temperature 102° F., pulse 120, breathing 24.

Over both lungs subcrepitant râles.

Sixth day: Temperature 101° to 99° F.

Seventh day: Temperature 100° to 99° F.

After this, temperature between 99° and 100° F. until the twelfth day.

She was put to bed, on milk diet, with no treatment.

Twelfth day: She was discharged cured.

CASE V. *Bronchitis with Asthma.*—Female, 26 years old, November 18, 1897.

For the last four years she has had an attack of asthma every fall. Each attack has been accompanied with bronchitis. Two days ago, after getting her feet wet, she awoke in the night with spasmodic asthma and cough with mucous expectoration. The dyspnœa has continued, with exacerbations at night. Now the temperature is 98° F., pulse 100, breathing 36. Over both lungs sibilant and sonorous breathing and subcrepitant râles.

While she was in the hospital she took nitroglycerin, emphysema mixture, ipecac, and the iodide of potash.

Fifteenth day: She was discharged, with no cough and no dyspnœa.

3. There are cases characterized by the production of very large quantities of mucus, pus, and serum from the bronchi. During each twenty-four hours the patients cough up as much as a pint of muco-purulent matter, and they continue to do so day after day. In these patients the course of the disease is unusually severe and protracted.

CASE VI. *Acute Bronchitis; Profuse Muco-Purulent Expectoration.*—Male, 54 years old, December 27, 1893.

On December 25 he had a chill, followed by fever, cough, dyspnœa, pain over the front of the chest, and diarrhœa.

Now the tongue is moist and clean; there is cough with profuse, white mucous sputum; urine 1021, a trace of albumin. All over the chest is exaggerated resonance and sibilant and sonorous breathing.

He is given one-fourth of a grain of ipecac every hour for six doses.

Fifth day: The sputum is still very profuse and now contains a good deal of pus.

Seventh day: The sputum is still very profuse and purulent; he is given five grains of potassium iodide and twenty minims of tinctura nucis vomicæ four times a day.

Ninth day: The sputum is still abundant, but not as purulent. He is put on the continuous inhalation of creosote.

Twelfth day: The man is out of bed, with very little cough or expectoration.

The temperature ran from 100° to 102° F., the pulse from 80 to 90, and the breathing from 20 to 24 up to the eleventh day.

Seventeenth day: There are no cough, no sputum, no physical signs, and the man is discharged cured.

CASE VII. *Acute Bronchitis, Protracted.*—Male, 25 years old, January 2, 1897.

Three weeks ago he began to feel chilly, to have cough, expectoration, at first mucous, afterward muco-purulent, with pain under the sternum and considerable dyspnœa. Now the color and nutrition are good, cough with muco-purulent expectoration. Over the left lung subcrepitant râles and diminished breathing.

The man was put upon the inhalation of creosote.

Thirty-eighth day: He was discharged nearly well.

(For chart of Case VII, see Fig. 27.)

4. The inflammation extends down to a great many of the small bronchi, so that they contain mucus and pus. This gives an abundant subcrepitant râle over the whole of both lungs. The act of breathing is seriously interfered with. In some of the patients there is an evident attempt to overcome the obstruction; they breathe with difficulty, but with a good deal of effort. In others, however, with an equal amount of obstruction, the breathing is quiet and feeble. This form of bronchitis is sometimes fatal.

CASE VIII. *Acute Bronchitis; Death.*—Female, 44 years old, February 26, 1898.

She has had several attacks of rheumatism.

She has had dyspnœa on exertion for the past five years. Four weeks ago she began to have cough with profuse yellow expectoration, and a good deal of dyspnœa. Now the color and nutrition are poor; there is a good deal of dyspnœa. Over both lungs sibilant breathing and coarse and subcrepitant râles.

She continued to get worse until the end of the fifth week of her attack, when she died.

Autopsy.—The body is well nourished.

In both lungs there is a large quantity of muco-pus in the bronchi. The lungs are emphysematous. There are a few old miliary tubercles in the upper and lower lobes of the right lung.

The heart: There is hypertrophy of the wall of the left ventricle, without dilatation.

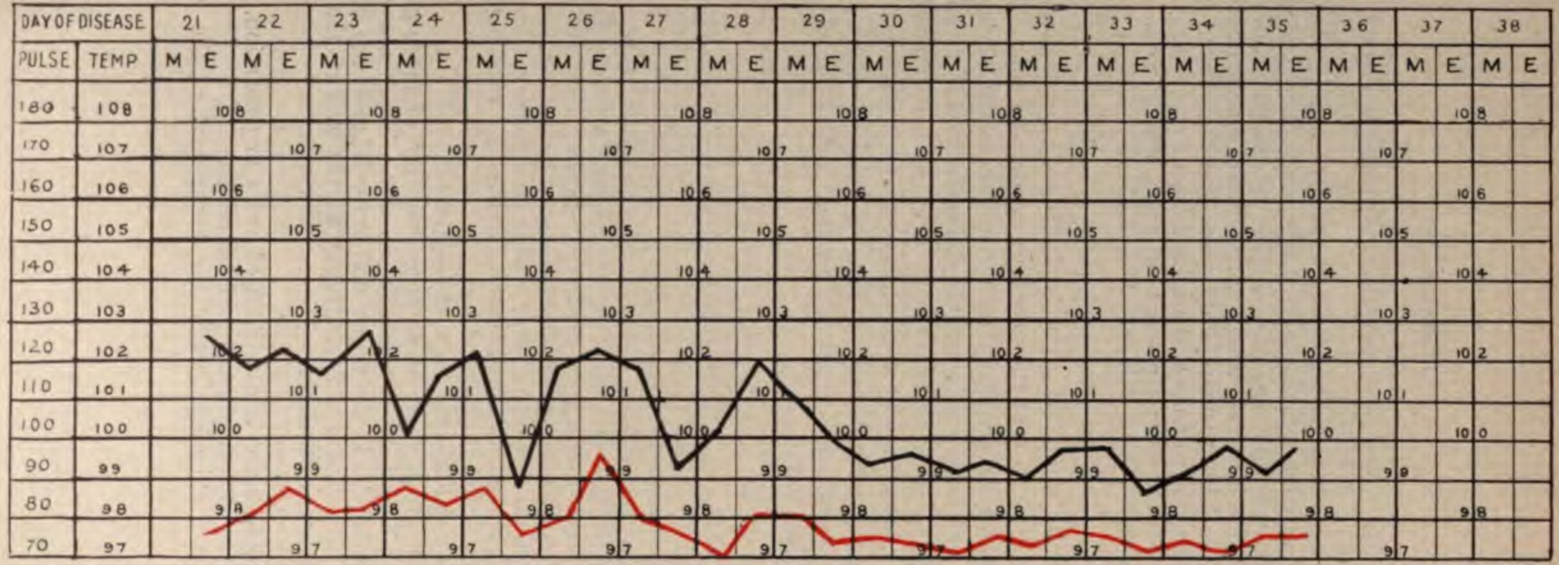
The spleen is soft.

The kidneys are in condition of acute degeneration.

The liver is covered with old adhesions.

There are old adhesions in the lower part of the peritoneal cavity.

FIGURE 27.



Temperature black; pulse red. Case 7

5. The bronchitis is mild, but the patients have high temperatures and seem quite ill because they have epidemic influenza.

CASE IX. *Acute Bronchitis; Influenza.*—Female, 26 years old, February 28, 1895.

One week ago she began to cough without expectoration. Two days ago began to have general bodily pains and headache.

Now her face is flushed, herpes on the lips, moderate prostration; urine 1026, a considerable quantity of albumin and granular casts; cough with mucous sputum. Over both lungs there is sibilant and sonorous breathing and coarse râles.

She gradually improved and was discharged cured on the twenty-seventh day.

(For chart of Case IX, see Fig. 28.)

6. In some of the cases there is an unusual congestion of the bronchi, with bleeding and transudation of serum. The sputum is abundant and contains a good deal of blood and serum. The difficulty in breathing is considerable, so that the patients may become cyanotic. There are a great many subcrepitant râles to be heard all over the chest. While the congestion continues the condition of these patients is often very alarming. They may die.

7. In many patients bronchial asthma is more distressing than the bronchitis. The patients suffer from all the discomforts which belong to the spasmodic dyspnœa.

CASE X. *Acute Bronchitis; Protracted Asthma.*—Female, 25 years old, November 24, 1894.

Three days ago she began to cough and to have dyspnœa. The sputum was bloody. Now the tongue is thickly coated; there is marked dyspnœa; there is cough with bloody expectoration.

Over both chests there is sibilant and sonorous breathing and subcrepitant râles.

Seventh day: The dyspnœa is much better.

The sputum is mucous, without blood.

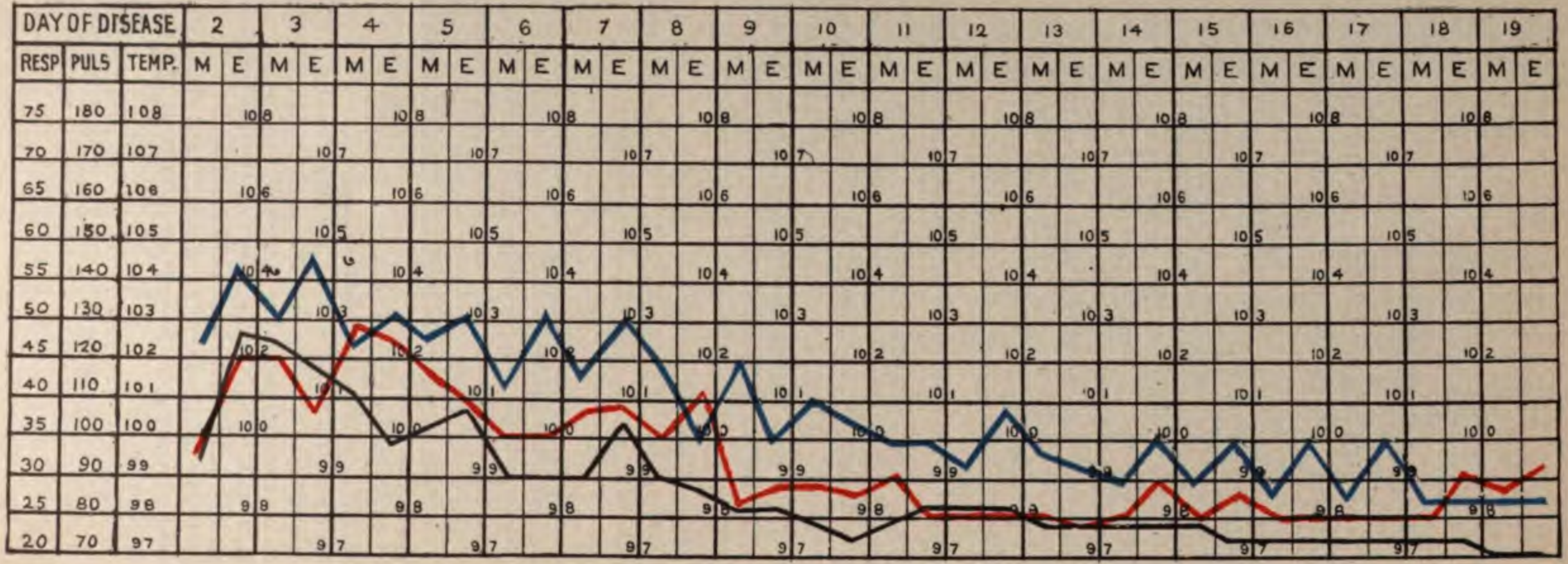
She continued to improve until the fifteenth day, when there was no more cough, no more sputum, and no more dyspnœa.

She was discharged from the hospital.

Two days after leaving the hospital the dyspnœa returned, with cough and scanty mucous expectoration.

Thirty-seventh day: She has marked asthmatic breathing, with sibilant and sonorous breathing over both lungs.

FIGURE 28.



Temperature black; pulse red; respiration blue. Case 9

Thirty-ninth day: The asthmatic breathing continues and she coughs up a little blood.

Forty-second day: The dyspnœa continues, with considerable muco-purulent and bloody sputum.

Seventy-fourth day: She has gradually improved, until there is no more asthma and but very little cough and expectoration.

CASE XI. *Acute Bronchitis; Asthma.*—Female, 32 years old, November 1, 1893.

Two weeks ago she began to have cough with muco-purulent expectoration.

A week later she began to have continuous dyspnœa, with exacerbations at night.

Now, temperature 99° F., pulse 112, breathing 24. Cough with mucous expectoration; marked dyspnœa. Sibilant and sonorous breathing over both lungs.

Eighteenth day: The cough and expectoration have diminished; she has no more asthma.

After this she had no more asthma; the cough and expectoration gradually diminished, and on the twenty-seventh day she was discharged cured.

8. In the course of the bronchitis there is developed a broncho-pneumonia. This, as a rule, is not extensive and results in the consolidation of only a small part of one lung. The area of hepatization is so small that the physical signs are not pronounced; a little dulness, broncho-vesicular breathing, and a louder voice are all that we can expect to get. The most noticeable feature of the clinical symptoms is that the patients do not get better of their bronchitis within the ordinary time, but continue to cough and have an afternoon fever.

CASE XII. *Acute Bronchitis; Broncho-Pneumonia.*—Female, 13 years old, September 20, 1893.

Two weeks ago she began to have cough, mucous sputum, pain in the left side, headache, and vomiting.

Two days later she went to bed.

One week ago she had a chill, followed by sweating, and began to lose flesh and strength pretty rapidly.

Now, temperature 99°, pulse 132, breathing 28. She is pale, has no appetite; the tongue is dry and coated; there is cough with mucous sputum. Over both lungs there is sibilant and sonorous breathing. Over the lower part of the left lung there is a circumscribed area where there is dulness on percussion and a crepitant râle.

The temperature continued not above 99° F., the pulse 80, the breathing 20.

The cough was no better until the twenty-second day; after that it improved, and by the twenty-sixth day there was no more bronchitis.

9. There are cases in which the cough, with muco-purulent sputum, an afternoon fever with sweating at night, and gradual loss of flesh and strength continue for weeks and months. These patients may become so enfeebled as to cause great anxiety. It is necessary to be very careful to exclude the presence of broncho-pneumonia and to examine the sputum repeatedly for tubercle bacilli.

CASE XIII. *Acute Bronchitis, Protracted.*—Female, 33 years old, June 6, 1895.

On March 21 she had an attack of influenza with bronchitis. Since that time she has continued to have cough with muco-purulent sputum and some dyspnœa on exertion. On one occasion there was a small hæmoptysis. She has lost twenty pounds in weight and is quite feeble.

Now, temperature 99° F., pulse 88, breathing 36.

There are no marked physical signs.

The sputum was examined several times for tubercle bacilli and none were found.

She continued to have cough and muco-purulent sputum, and finally left the hospital on July 5, in very much the same condition as when she entered it, nearly four months after the beginning of the bronchitis.

CASE XIV. *Acute Bronchitis, Protracted.*—Male, 35 years old, January 23, 1897.

Three months ago he caught cold and began to cough, with mucous sputum; he also had headache and night sweats. He continued to work until two days ago, but lost strength and was short of breath.

Now, temperature 102° F., pulse 80, breathing 24; harsh breathing and subcrepitant râles over both lungs.

No tubercle bacilli were found in the sputum.

He was put on inhalations of terebene and the internal use of hydriodic acid. He improved rapidly and was discharged cured on February 9, after a bronchitis of nearly four months' duration.

Treatment.—The mild cases of bronchitis easily get well without treatment; but the severe cases, the protracted cases, the cases with asthma, the cases with epidemic influenza, are very different. If left to themselves they continue indefinitely or even prove fatal; if they are properly treated they do very well.

In any severe bronchitis with congestion and œdema of the lungs, thorough dry-cupping of the whole chest should always be done.

The drugs which are of real service for bronchitis are: ipecac, potassium iodide, nux vomica, and creosote.

Those which control bronchial asthma are: nitroglycerin, chloral hydrate, potassium iodide, belladonna, and the fumes of stramonium and the nitrite of potassium.

These drugs can be given separately and in various combinations.

Ipecac is given by itself in doses of from one-tenth to one-quarter of a grain every hour, or combined as follows.

For a simple acute bronchitis of the larger and smaller bronchi the following formula will be found of service:

℞
Extracti belladonnæ.................................gr. $\frac{1}{40}$
Pulveris Doveri.......................................gr. $\frac{1}{40}$
Pulveris ipecacuanhæ...............................gr. $\frac{1}{10}$
Quininæ sulphatis....................................gr. $\frac{1}{4}$
One such pill or tablet every hour.

If the small bronchi are involved or if the bronchitis has continued for more than a week, the following formulæ are used:

℞
Extracti belladonnæ.................................gr. $\frac{1}{8}$
Pulveris ipecacuanhæ...............................gr. $\frac{1}{8}$
Potassii iodidi...grs. v
One such capsule every three hours.

℞

Morphinæ sulphatis	gr. $\frac{1}{30}$
Pulveris ipecacuanhæ	gr. $\frac{1}{6}$
Extracti nucis vomicæ	gr. $\frac{1}{6}$
Salol	gr. iii

One such pill every three hours.

℞

Potassii iodidi	ʒ ii
Tincturæ opii deodorati	ʒ i
Vini ipecacuanhæ	ʒ ii
Syrupi pruni Virginianæ	℥ ii
Aquæ	ad ℥ iv

In teaspoonful doses.

℞

Potassii iodidi	ʒ ii
Codeiæ	grs. v
Tincturæ sanguinariæ	ʒ ii
Syrupi pruni Virginianæ	℥ ii
Aquæ	ad ℥ iv

In teaspoonful doses.

℞

Pulveris ipecacuanhæ	gr. $\frac{1}{6}$
Potassii iodidi	grs. v
Pulveris Doveri	gr. $\frac{1}{12}$

One such capsule every three hours.

In the cases with excessive expectoration of mucus and pus, the continuous inhalation of creosote, and the internal use of tincture of nux vomica in doses of from ten to twenty minims every three hours, are of real service.

When there is marked congestion of the bronchi it is important to dry-cup the chest very thoroughly. There is also the indication to relieve the congestion of the lungs by increasing the rapidity of the circulation. The following formula is adapted for this purpose:

℞

Tincturæ nucis vomicæ	♏ lxxx
Extracti convallariæ fl	♏ lxxx
Extracti digitalis fl	♏ viii
Syrupi pruni Virginianæ	℥ i
Aquæ	ad ℥ ii

In teaspoonful doses.

Hypodermic injections of one-fiftieth of a grain of nitroglycerin sometimes have a very marked effect.

When there is bronchial asthma with the bronchitis we combine the drugs for the bronchitis with the drugs which dilate the bronchi. In doing this we must notice whether the production of mucus and pus from the bronchi is profuse or scanty, for it may be proper to try either to increase or diminish the production of mucus.

It is also necessary to notice whether the contractions of the right and left ventricles of the heart are sufficiently forcible, so that cardiac stimulants can be given if necessary. In this way we have to vary the combinations of drugs.

℞

Nitroglycerin	gr. $\frac{1}{50}$
Pulveris ipecacuanhæ	gr. $\frac{1}{10}$

To be given together every two hours.

℞

Chloral hydrate	ʒ i
Potassii iodidi	ʒ i
Vini ipecacuanhæ	ʒ ii
Tincturæ nucis vomicæ	ʒ i
Glycerin	℥ i
Aquæ	ad ℥ vi

Tablespoonful every three hours.

℞

Potassii iodidi	ʒ i
Tincturæ belladonnæ	ʒ i
Spiritus ætheris co	ʒ i
Magendie's Solution of Morphine	♏ xxiv
Syrupus sarsaparillæ	℥ i
Aquæ	ad ℥ vi

Tablespoonful every three hours.

℞

Potassii iodidi	ʒ i
Tincturæ nucis vomicæ	ʒ i
Glycerin	℥ i
Aquæ	ad ℥ vi

Tablespoonful every three hours—to diminish a muco-purulent sputum.

℞

Potassii iodidi	ʒ i
Extracti quebracho fl. (or extracti grindeliæ robustæ fl.)	ʒ i
Syrupus sarsaparillæ	℥ i
Aquæ ad	℥ vi

Tablespoonful every three hours—to increase the production of mucus.

℞

Chloral hydrate	ʒ i
Extracti digitalis fl.	♏ xxiv
Extracti convallariæ fl.	ʒ ii
Tincturæ nucis vomicæ	ʒ i
Glycerin	℥ i
Aquæ ad	℥ vi

Tablespoonful every three hours—with a feeble heart.

2. THE ACUTE CATARRHAL BRONCHITIS OF OLD PERSONS.

The lesions are the same as in the bronchitis of adults.

The symptoms are more severe in proportion to the extent of the bronchitis, and the disease is not as well borne as it is in adults. There are, from the first, much prostration, an irregular febrile movement, a rapid and feeble pulse, difficult and oppressed breathing, cough with mucous expectoration, restlessness, sleeplessness, sometimes delirium, loss of appetite, nausea, vomiting. There are coarse and subcrepitant râles over both lungs, or only over a portion of one lung. In some cases no râles can be heard.

An acute bronchitis in old persons is often alarming and sometimes fatal.

The treatment is the same as in adults; but the nursing is even more important, the difficulty in feeding the patients is greater, and stimulants are more likely to be necessary.

3. THE ACUTE CATARRHAL BRONCHITIS OF CHILDREN.

The lesions are the same as in adults: congestion and swelling of the mucous membrane, after a time an increased production of mucus, with some subsidence of the swelling. But as the lungs of children are smaller, as the bronchi constitute a relatively larger portion of the lung, as the bronchi are smaller and more easily occluded, as such an occlusion may even be complete with unaëration of portions of the lungs, so we find bronchitis in children to interfere seriously with the functions of breathing and to constitute a serious disease.

Symptoms.—An acute bronchitis may be preceded by coryza, pharyngitis, tonsillitis, or laryngitis; it may occur as a primary inflammation; or it may complicate measles, whooping cough, or one of the infectious diseases.

In the mild cases there is no fever; the children hardly feel sick; but they cough, and coarse râles can be heard over both lungs. The inflammation regularly runs its course and subsides within one or two weeks.

In the majority of these mild cases no treatment is necessary, not even for the cough. It is wise, however, to keep these patients in the house until the bronchitis has subsided, unless the weather is warm and good.

In the more severe cases the invasion of the inflammation may be marked by general convulsions. There is a well-marked febrile movement, the temperature higher in the afternoon, sometimes falling even to the normal in the morning. The pulse is rapid, but usually not feeble. The breathing is rapid, sometimes insufficient; it may be made worse for a time by distension of the stomach with food. There may be alternating restlessness and drowsiness. There are subcrepitant and coarse râles heard over both lungs, sometimes early, sometimes late in the disease.

The inflammation regularly runs its course within two weeks, and the patients recover. But they are often alarmingly ill for several days and may die from the disease.

In infants only a few weeks old rapid breathing, fever, and prostration are the only symptoms, and the disease proves fatal within a few days.

CASE XV. *Acute Bronchitis; Child.*—Male, 8 months old, January 24, 1897.

Three days ago the mother noticed that the child was feverish and coughing, with alternating drowsiness and restlessness. Yesterday the child vomited. Now the temperature is 101° F., pulse 128, breathing 44. The breathing is harsh over both lungs.

Fifth day: Temperature 101° to 99° F.

Sixth day: Temperature 102° to 100° F.

Seventh day: Temperature 100° F.

Eighth day: Temperature 100° to 99° F.

The child had no medicine but a few drops of paregoric.

January 31: Discharged cured.

CASE XVI. *Acute Bronchitis; Child.*—Male, 2½ years old, October 25, 1898.

Two nights ago the child was restless and complained of headache.

Last night he had high fever, with rapid breathing, and looked very ill.

Now the temperature is 103° F., pulse 140, breathing 56. The child looks very sick. There is some twitching of the arms. There is sibilant breathing, and subcrepitant râles over both lungs.

Sixth day: The temperature had fallen to 99° F., pulse was 120, the breathing was comfortable; the child was decidedly better.

Seventeenth day: The child was discharged well.

CASE XVII. *Acute Bronchitis; Chill; Convulsions.*—Male, 6 months old, March 5, 1895.

The child had a little cough for a few days, then yesterday general convulsions, vomiting, and diarrhœa.

Now the temperature is 101° F., pulse 150, breathing 56; coarse and subcrepitant râles over both lungs.

Third day: Temperature 101° to 100°F., pulse 140 to 130, breathing 50 to 40.

Fifth day: Temperature 99° F., pulse 126, breathing 38.

Seventh day: Discharged cured.

Treatment.—Young children do not need much treatment for bronchitis; the nursing is the principal thing.

Many cases do best without any drugs at all, but small doses of ipecac, aconite, and opium are sometimes of service.

4. CHRONIC CATARRHAL BRONCHITIS.

Chronic catarrhal bronchitis may from the first have the character of a chronic inflammation; it may follow one or more attacks of acute bronchitis; it may complicate gout, emphysema, chronic endocarditis, interstitial pneumonia, pleuritic adhesions, phthisis; it may be produced by the inhalation of irritating substances.

Lesions.—The mucous glands produce too much mucus; they may be hypertrophied; the walls of the bronchi are thickened or thinned; the lumen of the bronchi may be narrowed or dilated.

Symptoms.—1. The mild cases: In the patients belonging to this class the bronchitis hardly amounts to more than an annoyance. They have a cough, either with or without mucous sputum, and that is all. They require no treatment, but may be benefited by a suitable climate. Most of them have some emphysema, but it is of the form that gives no symptoms.

2. The more severe cases: Of these there are a good many. All of them have emphysema; some also have chronic nephritis or chronic endocarditis.

The symptoms come on in attacks, each attack lasting for several weeks. The attacks are mostly in winter, but they also occur in summer. During the attacks the patients are seriously ill.

They have cough with muco-purulent sputum, constant or spasmodic dyspnœa, a febrile movement, and considerable loss of flesh and strength. They require active treatment for the bronchitis and the asthma. It is a matter of great consequence that they should live in a climate where there is the least liability to the attacks.

Case XVIII. *Chronic Bronchitis; Asthma.*—Male, 50 years old, November 24, 1893.

For the last five years he has had asthmatic attacks, with cough and scanty mucous expectoration; these attacks have been frequently repeated, and any exertion brings on dyspnœa.

Four days ago he was suddenly attacked in the night with asthma. Since then he has had continuous dyspnœa, cough with scanty mucous sputum, fever and sweating.

Now there is considerable cyanosis of the face and fingers; the tongue is heavily coated; he has marked bronchial dyspnœa; temperature 102.4° F., pulse 120, breathing 24; urine 1020, a little albumin.

Over both lungs there is sibilant and sonorous breathing.

The man was treated with ipecac, chloral hydrate, potassium iodide, and nux vomica.

The temperature gradually fell, the sputum first became more abundant and then diminished, the dyspnœa disappeared, and by December 2 the attack was over.

Case XIX. *Chronic Bronchitis, Emphysema, and Chronic Endocarditis.*—Male, 50 years old, November 1, 1895.

Since 1878 the man has had several attacks of rheumatism. During the same time he has had cough with mucous expectoration, dyspnœa, palpitation and pain in the heart, and œdema of the ankles. These symptoms have been repeated from time to time, with a steady increase in their severity, the exacerbations regularly depending on attacks of rheumatism and of bronchitis. For the past three weeks all these symptoms have been worse. He now has cough with mucous expectoration, orthopnœa, and sleeplessness.

Temperature 99° F., pulse 108, breathing 32.

There is sibilant and sonorous breathing and coarse râles over both lungs.

The heart is enlarged; its action is irregular; there is a mitral systolic murmur.

No tubercle bacilli were found in the sputum. His temperatures ran between 99° and 103° F. While the man was in the hospital the chest was dry-cupped; he took chloral hydrate, nitroglycerin, iodide of potash, tincture of nux vomica, ipecac, digitalis, convallaria, and inhalations of creosote.

The cough and expectoration continued until November 29. After that he improved and was discharged without any symptoms on December 10.

Case XX. *Chronic Bronchitis; Death.*—Male, 71 years old, November 12, 1894.

He has had cough with expectoration, some dyspnœa, and gradual loss of flesh for eleven years.

Two weeks ago the cough became worse, with muco-purulent sputum; temperature 99° F., pulse 120, breathing 50; urine 1025, a little albumin.

There is sibilant breathing and subcrepitant râles over both lungs.

November 18: Influenza bacilli were found in the sputum.

He continued to have cough and muco-purulent sputum; the temperature was not above 99° F., but he gradually grew weaker and died on December 24.

Autopsy.—The left lung is firmly attached to the wall of the chest by old adhesions.

At the apex there are some old pigmented, fibrous nodules. In both lungs the bronchi are full of pus.

There is old thickening of the mitral and tricuspid valves, with fatty degeneration of the heart muscle.

The liver is cirrhotic.

The kidneys show the lesions of chronic nephritis.

3. There are cases of chronic bronchitis with very large quantities of muco-purulent sputum, without dilatation of the bronchi, which continue to get worse for years. The bacteriology of these cases needs to be worked out.

Their drug treatment is very unsatisfactory; a dry, elevated inland climate may do them some good.

Case XXI.—Male, 47 years old, November 12, 1900.

The man says that he has had dyspnœa on exertion all his life, but no asthmatic attacks. Two years ago he began to cough and expectorate large quantities of muco-purulent matter. He has had three small hæmoptyses and has gradually lost flesh and strength. The dyspnœa has been worse since the cough began. He has been unable to work for six months.

He now weighs 141 pounds. There are no physical signs, except those of pulmonary emphysema. The hæmoglobin is 85 per cent, the red blood cells 5,024,000, the white blood cells 8,000.

While he was in the hospital he lost six pounds in weight; the temperature varied from 99° to 101° F.; the quantity of sputum was from fifteen to twenty-three ounces in each twenty-four hours. The only micro-organism that could be found in the sputum was the bacillus pyocyaneus.

The man left the hospital unimproved.

4. There are cases of chronic bronchitis with interstitial pneumonia which go on for years. The interstitial pneumonia is usually secondary to pleurisy or to broncho-pneumonia.

The principal indication for treatment in these patients is to find a suitable climate.

5. There are cases of chronic bronchitis with considerable dilatation of the bronchi. The most marked feature of these cases is the way in which they raise the sputum. They have

attacks of coughing at intervals of several hours, and then bring up as much as a pint of sputum.

Between the attacks there is little or no cough or sputum.

The treatment is unsatisfactory.

LECTURES
ON
THE PRACTICE OF MEDICINE
WITH CASES AND CHARTS
BY
FRANCIS DELAFIELD, M.D., LL.D. (Yale)

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PNEUMONIA.

The term Pneumonia, or Pneumonitis, is employed to designate the inflammations of the parenchyma of the lungs as distinguished from those of the bronchi and the pleura. By the parenchyma of the lungs we mean the air vesicles, the air passages, and the smallest bronchi.

There are a number of different forms of pneumonia, distinguished from each other by their causes, their lesions, and their symptoms. In the present state of our knowledge we cannot make a scientific classification of the different forms, but have to be contented to describe the different varieties of pneumonia under arbitrary names.

We distinguish, therefore, primary lobar pneumonia, secondary lobar pneumonia, lobar pneumonia with the formation of new connective tissue, broncho-pneumonia, pneumonia of heart disease, interstitial pneumonia, tubercular pneumonia, syphilitic pneumonia.

We can also make a classification according to the method of infection, and speak of pneumococcus pneumonia, streptococcus pneumonia, influenza pneumonia, tubercular pneumonia, syphilitic pneumonia.

PRIMARY LOBAR PNEUMONIA.

Definition.—An acute exudative inflammation of the parenchyma of the lung accompanied by the growth of pneumococci or streptococci in the exudate.

Nature of the Disease.—Much has been said and written as to whether lobar pneumonia is an inflammation of the lungs or an infectious disease. At the present time our knowledge of the disease is more definite.

The ordinary cases of lobar pneumonia are examples of a pneumococcus infection with inflammation of the lungs. The infection and inflammation may be confined to the lungs; they often involve the pleura; they may involve the pericardium, the endocardium, the pia mater, the joints, and the peritoneum.

States wholly or in great part above the 39th parallel.	Per 1,000 deaths.	Per 1,000 inhabitants.	States wholly or in great part below the 39th parallel.	Per 1,000 deaths.	Per 1,000 inhabitants.
1. Maine	51.22	0.62	1. Delaware	56.41	0.68
2. New Hampshire.	62.46	0.96	2. Maryland	59.59	0.70
3. Vermont	58.58	0.59	3. Virginia; W Virginia	75.66	0.94
4. Massachusetts	56.29	0.98	4. North Carolina	71.34	0.80
5. Rhode Island	58.23	0.78	5. South Carolina	102.58	1.26
6. Connecticut	55.96	0.72	6. Georgia	99.54	1.17
7. New York	60.55	0.87	7. Florida	113.55	1.39
8. New Jersey	51.61	0.59	8. Alabama	123.81	1.47
9. Pennsylvania	44.84	0.58	9. Mississippi	127.21	1.68
10. Ohio	60.27	0.65	10. Louisiana	94.15	1.75
11. Indiana	80.66	0.88	11. Texas	105.43	1.58
12. Illinois	77.94	0.96	12. Arkansas	183.42	2.98
13. Michigan	69.64	0.67	13 Kansas	112.13	1.49
14. Wisconsin	54.55	0.50	14. Kentucky	78.49	0.95
15. Minnesota	55.30	0.39	15. Tennessee	84.03	1.04
16. Iowa	75.32	0.61	16. Missouri	103.96	1.41
17. Nebraska	87.30	0.93	17. Nevada	81.30	1.18
18. Oregon	55.76	0.34	18 California	46.77	0.65
19. Colorado	50.67	0.48	19. District of Columbia	60.87	0.98
Average	61.43	0.69	Average	93.70	1.27
			Excess over Northern States	32.27	0.58

Pneumococci are also sometimes found in the blood in very severe cases. The pneumococcus toxin may be mild or virulent.

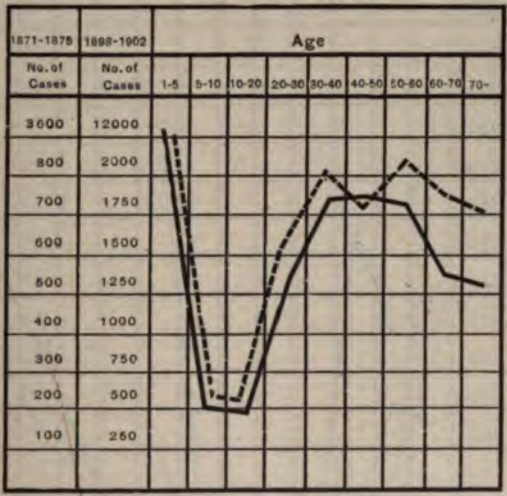
The point that is not settled is whether the pneumococci are inhaled into a healthy lung, act as direct irritants, and cause the inflammation, or whether the lung must first be inflamed from other causes so as to be a favorable soil for the growth of the pneumococci.

When influenza is prevalent the pneumococcus infection is often complicated by an infection with the influenza bacillus. The influenza bacillus causes unusually severe inflammations of the bronchi complicating the pneumonia, and the patients are poisoned by the toxins of both micro-organisms.

In dirty rooms, hospital wards, asylums, and prisons streptococci are often present in great numbers and take the place of pneumococci in causing lobar pneumonia. If the lungs are in the condition of venous congestion from any cause, this infection is particularly apt to take place.

Etiology.—Lobar pneumonia is a very widely distributed disease. There are few countries in which it does not prevail, the mortality ranging from 1.10 to 2.30 per cent for each 1,000 inhabitants. In the United States, according to the eighth and ninth census reports, the disease is of more frequent occurrence in the South than in the North. (See table on opposite page.)

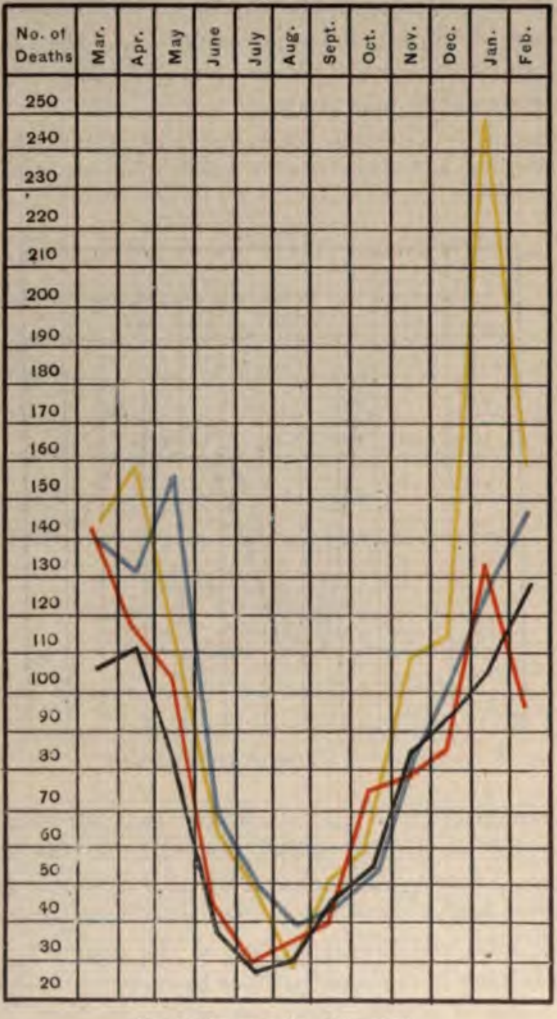
FIG. 29



Number of deaths at different ages.

In New York pneumonia is one of the ordinary diseases—broncho-pneumonia in children, lobar pneumonia in adults. The whole number of deaths from pneumonia in New York from March 1, 1871, to February 28, 1875, was 7,873. The number of deaths at different ages is shown by the solid line in Figure 29. The whole number of deaths from pneumonia from March 1, 1898, to February 28, 1902, was 24,228. The number of deaths at different ages is shown by the broken line in Figure 29.

FIG. 30

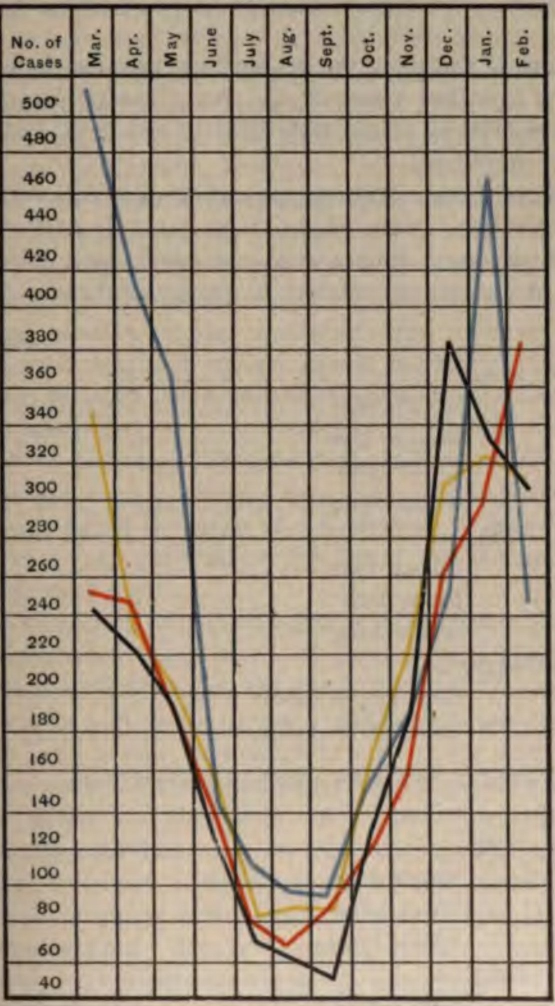


Deaths by months, 1871-1875.

It will be seen that the number of deaths in children from 1 to 5 years old is very large; these are mostly broncho-pneumonias.

Most of the deaths from lobar pneumonia are between the ages of 30 and 60.

FIG. 31



Deaths by months, 1898-1902.

In New York the disease prevails in the winter and spring, with but few cases in the summer. Generally the curves of mortality correspond pretty closely to the curves of temperature

—the colder the weather the greater the number of deaths from pneumonia. In Figure 30 are given the deaths from pneumonia by months, in persons over five years of age, for four years, 1871-1872 in black, 1872-1873 in red, 1873-1874 in blue, 1874-1875 in yellow.

In Figure 31 are given the deaths from pneumonia, by months. in persons over five years of age, for a record period of four years, 1898-1899 in black, 1899-1900 in red, 1900-1901 in blue, 1901-1902 in yellow.

Morbid Anatomy.—The inflammation, as a rule, involves the whole of one lobe, or the whole of one lung, or portions of both lungs. Juergensen, from a study of 6,666 cases, gives the following table to show the relative frequency of the situation of the lesion:

	PER CENT.
Right lung	53.70
" " upper lobe	12.15
" " middle lobe	1.77
" " lower lobe	22.14
" " whole lung	9.35
Left lung	38.23
" " upper lobe	6.96
" " lower lobe	22.73
" " whole lung	8.54
Both lungs	8.07
" " both upper lobes	1.09
" " both lower lobes	3.34

I have found in 359 cases observed in New York:

	CASES.
Right lung	183
" " upper lobe	51
" " lower lobe	90
" " whole lung	42
Left lung	123
" " upper lobe	16
" " lower lobe	87
" " whole lung	20
Both lungs	53

The inflammation in acute lobar pneumonia is of pure exudative type, characterized by congestion, emigration of white blood cells, diapedesis of red blood cells, and exudation of blood plasma, while the tissue of the lung remains unchanged. For clinical purposes it is important to have as distinct an idea as possible of the condition of the lung while it is the seat of such an exudative inflammation, so that we describe the condition in which the lung is found while the inflammation is going through its regular stages of congestion, exudation, and resolution.

During the first hours of the inflammation only irregular portions of the lobe which is to be inflamed are involved; later, the entire lobe. The lung is congested, œdematous, tough, but not consolidated. The air spaces contain granular matter, fibrin, pus cells, red blood cells, and epithelial cells. The epithelium remaining on the walls of the air spaces is swollen; there are large numbers of white blood cells in the capillaries. The larger bronchi are congested, dry, or coated with mucus; the small bronchi contain the same inflammatory products as do the air spaces. The pulmonary pleura, as a rule, is not coated with fibrin. This is called the stage of "congestion." The stage of congestion, as a rule, only lasts a few hours, but it may be protracted for several days.

When the exudation of the inflammatory products has reached its full development, the presence of these products within the air spaces and bronchi causes the lung to be solid, and at this time the lung is said to be in the condition of "red hepatization." The lung is now consolidated, red; its cut section looks granular, the granules corresponding to the plugs of inflammatory matter within the air spaces. For some time after death the inflammatory products remain solid and the cut section of the lung dry; but later, with the commencement of postmortem changes, these products soften and the cut section is covered with a grumous fluid. The air vesicles, the air passages, the small bronchi, and sometimes the large bronchi, are filled and distended with fibrin, pus cells, red blood cells, and epithelium. In spite of the pressure on the walls of the air spaces the blood vessels in their walls remain pervious. The pulmonary pleura is coated with fibrin and the interstitial connective tissue of the lung is infiltrated with the same substance. The hepatized lobe is increased in size, sometimes so much so as to compress the rest of the lung. About one-fourth of the fatal cases die in the stage of red hepatization, at any time from twenty-four hours to eleven days after the initial chill.

After the air spaces have become completely filled with the exudate, if the patients continue to live, there follows a period during which the exudate becomes first decolorized and then degenerated. This is the period of "gray hepatization." The lung remains solid; its color changes, first to a mottled red and gray, then to a uniform gray. The coloring matter is discharged from the red blood cells and the exudate begins to degenerate and soften. The lung is found passing from red to gray hepatization at any time between the second and the eighteenth day of the disease. It is found completely gray at any time from the fourth to the twenty-fifth day. About one-half of the cases die in the condition of mottled red and gray hepatization; about one-fourth in the condition of gray hepatization.

If the patients recover, the exudate undergoes still further degeneration and softening, and is removed by the lymphatics. This is the stage of "resolution." It should commence immediately after defervescence and be completed within a few days. But it may not begin until a number of days after defervescence, or it may be unusually protracted.

Modifications of the Inflammation.—The lung, instead of being freed from the exudate at the regular time, may remain in the condition of gray hepatization for weeks.

The quantity of inflammatory products may be so great that the blood vessels are compressed and portions of the lung become necrotic. To this necrotic condition may be added a very considerable production of leucocytes, and in this way abscesses are formed in the lung.

In a very few cases a hepatized lung is infected with the bacteria of putrefaction and the patients have gangrene of the lung.

The ordinary pleurisy of pneumonia only coats the pleura with fibrin. But there are cases with very considerable exudations of either clear or purulent serum.

In the cases in which there is a double infection with the influenza bacillus and with pneumococci, there is often an intense general catarrhal bronchitis with a large production of muco-pus, and in some cases an excessive congestion of the lung with comparatively little hepatization.

The lymphatic vessels in the pulmonary pleura and in the septa between the lobules may be filled with pus cells, and the pleura and the septa infiltrated with fibrin and pus.

It is often stated that lobar pneumonia can be changed into a tubercular pneumonia or a chronic pneumonia, but I believe that the cases thus described are really examples of pneumonias which were of tubercular or of productive character from the very outset.

The pia mater is sometimes the seat of an acute exudative inflammation with serum, fibrin and pus, and pneumococci in the pus cells.

The pericardium may be the seat of acute exudative inflammation with fibrin, pus, and pneumococci.

The aortic and mitral valves may be the seat of ulcerative endocarditis with pneumococci. This usually comes on after the pneumonia.

An acute peritonitis is one of the rare complications.

Purulent inflammation of the knee joints with a pneumococcus infection is seen in a few cases.

The regular condition of the kidneys is that of acute degeneration of mild type, without any renal symptoms except the presence of small quantities of albumin and a few casts in the urine. Sometimes, however, there is an acute exudative nephritis, especially if the pneumonia occurs with influenza.

Pneumococci are sometimes found in the blood during the life of the patient.

Symptoms. Physical Signs.—During the stage of congestion the lung is more dense than normal, but is not consolidated; the bronchi and some of the air spaces contain inflammatory products; the pleura is not yet coated with fibrin. The percussion note, therefore, remains unchanged or its pitch becomes higher, its duration shorter, and its quality less distinctly pulmonary. The respiratory murmur is either rude or diminished in intensity. The inflammatory products in the small bronchi may give a subcrepitant râle. If the larger bronchi are also inflamed there may be coarse râles and sibilant and sonorous breathing. As there is no fibrin yet on the pleura, there is no crepitant râle. It is evident, therefore, that during this stage of a pneumonia we must expect that the physical signs will either not be very marked or else absent altogether.

During the stages of red and gray hepatization the air spaces and small bronchi are filled with inflammatory products and are impervious to air. The larger bronchi are coated with mucus or filled with fibrin. But, although in all cases the lung is consolidated, there is a good deal of difference as to the quantity of inflammatory products, the size of the consolidated lobe, the closeness with which its surface is applied to the chest wall, and the degree of motion of which it is capable. The pulmonary pleura is coated with fibrin; occasionally there is serum in the pleural cavity. The percussion sound, therefore, is more or less dull, or flat, or tympanitic, or of cracked-pot quality. Any considerable quantity of fluid in the pleural cavity gives flatness.

A lobe of which the air spaces are distended with exudation, so that the lobe is increased in size and its surface pressed closely against the wall of the chest, gives either marked dulness, or flatness, or tympanitic resonance, or, in young persons, the cracked-pot sound.

The dulness on percussion is less marked, or is even absent altogether, if the inflamed lobe is very much congested but contains little exudate; if the quantity of exudate is not sufficient to distend the air spaces and increase the size of the lobe; if the area of consolidation is small or is situated in the central portions of the lung; or if the ribs have undergone the senile changes which cause them to give increased resonance. It is especially in old persons that these reasons for the absence of dulness on percussion often exist.

The vocal fremitus is, as a rule, increased over the consolidated lung. There is no satisfactory reason why this should not always be the case, but as exceptional conditions we find the vocal fremitus diminished or absent.

Bronchial breathing and bronchophony should be heard over the consolidated lung, but we may get bronchophony without bronchial breathing, or we may get neither bronchophony nor bronchial breathing. The absence of bronchophony seems to be due sometimes to the incomplete consolidation, sometimes to the occlusion of the large bronchi. The absence of bronchial breathing may be due to occlusion of the large bronchi or to the absence of movement of the lung. As the crepitant râle is due to the friction of the fibrin on the surface of the pleura, the râle will not be produced unless fibrin is present and the lung capable of movement. So we find in different cases a great difference as to the presence or absence of the crepitant râle. In some cases we only get it after coughing or with a forced inspiration; in some cases it is only heard at the beginning of red hepatization while the lung still moves; in some cases it is heard throughout the stages of red and gray hepatization; and in some cases it is absent altogether.

In the stage of resolution the products of inflammation are softened and rapidly absorbed, the air re-enters the small bronchi and air spaces, the lung moves more and more freely. So with the increased motion of the lung we get the crepitant râle due to the friction of the pleura. With the softening of the inflammatory products we get the subcrepitant and coarse râles in the bronchi. The bronchial breathing and bronchophony disappear, if they have been present. Normal vesicular breathing becomes more and more distinct. The percussion note loses its dull or flat or tympanitic or cracked-pot quality, and approaches nearer and nearer to the normal, but yet the changes in the percussion note last the longest of all the physical signs, and even long after complete resolution some dulness is often present.

✓ Resolut

It is always to be remembered that it is in old persons that the physical signs are the least constant and the least well marked.

Rational Symptoms.—In from one-sixth to one-third of the cases there are prodromic symptoms. Chilliness, a little fever, general malaise, and feelings of oppression about the chest continue for from one to four days. These symptoms probably correspond to a protracted period of congestion.

In about 90 per cent of the cases there are, during the first twenty-four hours, one or more decided chills, and it is from the time of the chill that we count the days of the disease.

The temperature rises at once and reaches it maximum by the afternoon of the first, second, or third day, but very often the highest temperature of the disease will be reached during the twenty-four hours preceding defervescence. An afternoon temperature of 104° and a morning temperature of 102° or 103° F. are about the normal temperatures of a lobar pneumonia. A sudden rise of temperature during the course of the disease indicates the extension of the pneumonia or the development of a complication. The removal of the patient from one house to another is, as a rule, followed by a rise of temperature. In persons not suffering from malarial poisoning and not taking antipyretics there are quite often irregular rises and falls of several degrees of temperature which we cannot account for. Pneumonias involving the upper lobes usually have high temperatures. The height of the temperature is usually in proportion to the severity of the disease, but patients may get worse with a falling temperature, or may die with temperatures below the normal, or, very rarely, have no rise of temperature throughout the disease.

✓ Defervescence is usually by crisis, the fall of temperature beginning in the evening and reaching the normal or lower within from six to forty-eight hours. Occasionally a rapid defervescence with a fall of temperature to 97° or 96° is attended with so much prostration and such a feeble heart action that the condition of the patient is alarming. It is said that epistaxis, hæmaturia, or hemorrhage from the bowels may accompany defervescence.

In a considerable number of cases the fall of temperature is slow, extending over several days before it reaches the normal. In the cases which I have seen the time required for this slow defervescence has been from three to twelve days. Such a slow defervescence belongs especially to the cases complicated with influenza.

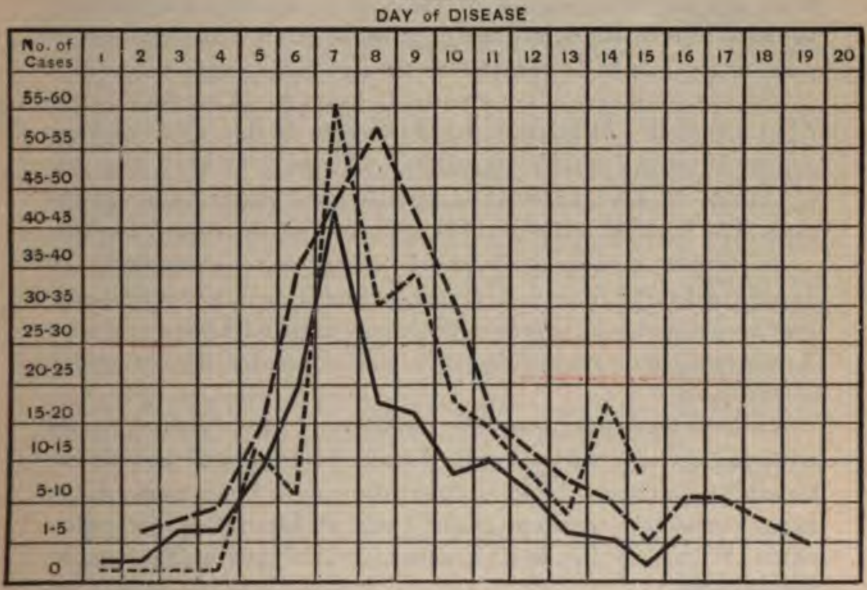
A rise of temperature after several days of partial or complete defervescence usually means pleurisy with effusion or empyema, but it may indicate influenza, a fresh pneumonia, abscess of the lung, or gangrene of the lung.

In Figure 32 are given the days on which the disease terminated by crisis.

I have the records of 21 cases which terminated by lysis and 131 cases which terminated by crisis. On the chart the curve of the crisis cases is given in the continuous black line. The cases were observed in the years 1894-1898.

Dr. Alfred Meyer gives the record of cases in the Mt. Sinai Hospital occurring during the ten years ending January 1, 1898. He found that 63 cases terminated by lysis and 200 by crisis. The curve of the crisis cases is given in the dotted line.

FIG. 32



Day of crisis.

Dr. Norris gives the record of cases in the Pennsylvania Hospital from May, 1897, to March, 1901. He found that 74 cases terminated by lysis and 301 by crisis. The curve of the crisis cases is given in the broken line.

The chart shows that the experience of three independent observers is practically the same—that the large majority of cases terminate by crisis on the seventh or eighth day of the disease. ✓

The condition of the heart's action and that of the pulse are

Pulse

of great importance. In a favorable case the pulse ought to be about 100 to the minute and fairly full. A pulse of over 120 is always cause for anxiety. The liability to failure of the heart's action, either gradual or sudden, constitutes one of the greatest dangers of pneumonia. It is not certain what the cause of the heart failure is, but it seems probable that it is due to the effects of the poison produced by the pathogenic bacteria of the disease.

The character of the heart failure resembles that of diphtheria: either the pulse gradually becomes more rapid and feeble, or there is a sudden cessation of the heart's action. The liability to heart failure seems ordinarily to begin on the third day of the pneumonia, and to be greatest on the day before defervescence.

The failure may involve the whole heart or be confined to the right ventricle. It seems to be the failure of the right ventricle which causes the venous congestion and œdema of the lungs. It is curious in some cases to observe a good radial pulse at the same time with the evidences of the failure of the right ventricle.

In persons already suffering from chronic endocarditis the danger of heart failure is very great; and it must be remembered that an old aortic or mitral stenosis may exist without a murmur. A complicating pericarditis, as a rule, increases the frequency of the pulse.

As the disease progresses the character of the breathing varies with the severity of the case. Rapid, labored, and insufficient breathing indicates either inflammation of a large part of the lung, excessive congestion of the lung, an intense general bronchitis, failure of the heart's action, or an intense pleurisy or pericarditis.

Cough may be developed as one of the first symptoms, or come on at any time in the disease, or be deferred until resolution has commenced. In old persons the cough is often slight or absent altogether.

The characteristic sputum of pneumonia is in the form of little, rounded, viscid pellets of red, yellow, or brownish color, mixed with thin, fluid mucus—the so-called rusty sputum. In rare cases, with a severe invasion, the patients may cough up a little pure blood at the beginning of the disease. In the bad cases the sputum is changed and the patients cough up considerable quantities of thin, dark-colored fluid. In some cases, throughout the disease there will be nothing but a little white mucus coughed up from time to time. In cases with an excessive catarrhal bronchitis the quantity of expectoration may be very large and like that of an ordinary bronchitis. It is by no means unusual, especially in old people, for the expectoration to be absent altogether. In some cases they cough up considerable quantities of tenacious mucus streaked with blood, or with blood diffused through it.

The sputum regularly contains red blood cells, leucocytes, considerable numbers of pneumococci in the leucocytes, or free, and sometimes influenza bacilli.

Pain

Pain over the inflamed lung, referred to the region below the nipple, is developed within twelve hours after the initial chill in the majority of cases, and after three or four days gradually disappears. This pain is sometimes so intense as to be for a time the principal symptom. In some persons there is no pain until resolution commences and the cough becomes troublesome. In old persons there is often not only an absence of pain, but there are no abnormal sensations whatever in the chest.

The appearance of the face is characteristic. There is a deep red flush about the centre of each cheek, and the expression of the face is a curious mixture of anxiety and apathy.

The skin may be either hot or dry, or bathed in perspiration throughout the disease.

The tongue is coated with a white fur, but in the favorable cases remains moist. A dry tongue indicates a severe form of the disease.

Headache, restlessness, and sleeplessness are troublesome during the first days of the disease.

There are three different kinds of delirium which may occur in the course of lobar pneumonia. I think it is important to distinguish between them.

Del.

First, there is a delirium of the same type that is seen with all the infectious diseases. It depends on the poison of the pneumonia, and varies in its severity with the virulence of the poisoning. It is usually associated with high temperatures and regularly indicates a severe form of the disease.

Second, there is an alcoholic delirium which belongs to persons who have been drinking up to the time of the invasion of the pneumonia. This delirium may easily pass into an attack of delirium tremens.

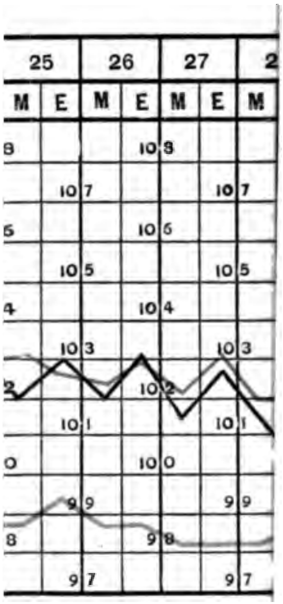
Third, there is a form of delirium to which I desire to call especial attention. It occurs both in mild and severe cases. It comes on at about the time of defervescence, or soon afterward. It may be accompanied by a fresh rise of temperature. It has more of the characters of a temporary insanity than of an ordinary delirium. It is not dangerous to life, but may last for as long as two weeks after defervescence. It often looks badly, but usually does no great harm, except in elderly people. They may remain permanently insane after recovering from the pneumonia.

Vomiting and diarrhœa are often seen during the first days of the disease.

It is of great importance to appreciate exactly the condition of the kidneys. The ordinary condition is a mild form of acute degeneration without symptoms, except a little albumin and a few casts in the urine. The kidneys soon return to their normal condition. In influenza pneumonias, however, there may be an acute exudative nephritis with a good deal of albumin and even blood in the urine. This seems to be due rather to the influenza than to the pneumonia. If the patients recover from the pneumonia the kidneys return to their normal condition.

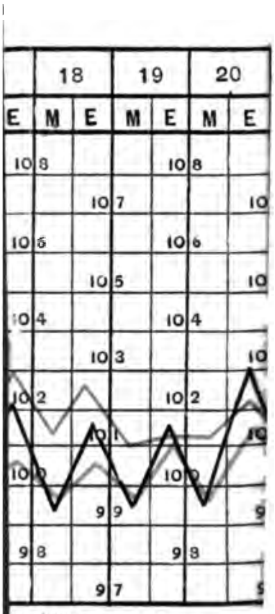
Persons already suffering from chronic nephritis, when they have pneumonia regularly have an exacerbation of the nephritis, which is apt to be serious and may cause death. It may very well happen that the existence of such a chronic nephritis has not been suspected until the exacerbation is brought out by the pneumonia.

Complications.—At any time in the course of a pneumonia, or after defervescence, there may be developed pleurisy with effusion or empyema on the same side as the pneumonia. The ordinary course is for the patient to get to the sixth or seventh day of his pneumonia and behave as if he were about to get well, but yet without complete defervescence. After a few days the temperature rises, higher in the afternoon, lower in the morning. With pleurisy with effusion the temperature does not



blue.

CRISY.



on blue.

SS OF LUNG.

usually get above 103° F.; with empyema it may be higher. The fluid may be in the lower part of the pleural cavity or be sacculated. The physical signs are the ordinary ones of fluid in the pleural cavity, but very often bronchial voice and breathing are to be heard over the fluid. This condition is of such common occurrence that it ought always to be recognized. But it habitually causes errors in diagnosis of the most serious character. The patients are supposed to have delayed resolution, chronic pneumonia, or tuberculosis.

CASE I. *Lobar Pneumonia followed by Pleurisy with Effusion.*—Male, 32 years old, December 19, 1896.

About a week ago he began to have headache, general pains, a little fever, and no appetite (influenza).

Yesterday, in the afternoon, he was seized with severe pain in the right side of the chest, with cough and higher fever.

Now, on the second day, over the lower half of the right chest in front and behind there is dulness on percussion, bronchial voice and breathing, and subcrepitant râles. Urine 1028, a trace of albumin.

Eighth day: Over the right side behind, from the midscapular region down, there was dulness on percussion and bronchial voice and breathing.

Eleventh day: There was flatness on percussion and bronchial voice and breathing over the whole right chest behind.

Fourteenth day: 810 c. c. of turbid serum were drawn off by the aspirator from the right chest.

Nineteenth day: 80 c. c. of serum were aspirated from the right chest.

Forty-third day: There was no fluid in the right chest and no consolidation of the lung.

Forty-fifth day: He was discharged cured.

(For Chart of Case I, see Fig. 33.)

There are rare cases in which lobar pneumonia is followed by abscess of the lung. The following history is an example:

CASE II.—A man, 24 years old, had his initial chill on April 18. The next day the temperature was up to 104° F., the pulse 134, the breathing 52. On the seventh day the physical signs of consolidation of the right lower lobe were evident, and on that day the temperature was 105°. On the fifth day the temperature began to fall, and continued to do so on the subsequent days until, on the ninth day of the disease, it reached the normal. There was a corresponding improvement in the whole condition of the patient, and he began to eat solid food. He continued to do well until the fifteenth day, when the afternoon temperature rose to 101.6° F., although the patient said he felt well and had a good appetite. On the sixteenth and seventeenth days the afternoon temperatures reached 102° F. There were dulness and diminished breathing over the right lower lobe; the patient was coughing up large quantities of muco-pus tinged with blood. On the twenty-first day the dulness persisted over the right lower lobe, but there were numerous coarse and subcrepitant râles. On the twenty-seventh day the patient coughed up a pint of pus, and on the next day he again coughed up a pint of pus. On the twenty-ninth day, over the right lower lobe behind there were only a little dulness and diminished breathing, but in front from the fifth rib down there was flatness. On the thirty-seventh day the sixth rib was resected near the sternum and the abscess in the lung opened. After this recovery was uninterrupted.

(For Chart of Case II, see Fig. 34.)

There are rare cases in which pneumonia is followed by gangrene of the lung. The following history is an example:

CASE III. *Lobar Pneumonia; Gangrene of the Lung.*—Male, 34 years old, January 23, 1893.

Three days ago in the evening, after exposure, he had a shaking chill and pains all over the body. The next morning his bowels were loose and he had an urgent dyspnœa.

Now, on the fourth day, his face was flushed, his tongue coated, and he had great difficulty in breathing; temperature 104.8° F., pulse 112, breathing 32; urine 1020, a little albumin and a good many casts. Over the lower two-thirds of the left lung behind there were diminished breathing, dulness on percussion, and a crepitant râle. The heart's action was regular; there was a systolic murmur at the apex.

Fifth day: He was actively delirious; temperature 103.4°-103.4° F., pulse 106-120, breathing 40-48. Over the left lung behind there was flatness on percussion. The sputum was dark-colored and foul-smelling.

Sixth day: He died.

Autopsy.—The right lung is congested; the lower lobe of the left lung is coated with fibrin, it is completely consolidated in the state of red and gray hepatization. In the lower part of this lobe there is an irregular gangrenous area.

The pericardium is coated with fibrin. The left ventricle of the heart is dilated and hypertrophied; the aortic valves are thickened; the mitral valves are thickened and coated with soft vegetations.

The spleen is large.

The liver is large and congested.

The kidneys are large, with adherent capsules, pale cortex, and cysts.

There are cases in which the quantity of fibrin is so large that portions of the lung become necrotic, but without gangrene or suppuration.

CASE IV. *Lobar Pneumonia; Necrosis of Areas of Lung.*—Female, 25 years old, January 11, 1875.

The woman was brought to the hospital very drunk; it was said that she had a chill three days before.

Fifth day: Over the right upper lobe there was dulness on percussion and bronchial voice and breathing; over the left upper lobe there were the same physical signs.

Seventh day: The woman seemed better in the morning, but became delirious in the evening. Over the right upper lobe there were coarse and gurgling râles.

Tenth day: She died.

Autopsy.—The right pulmonary pleura is coated with fibrin and pus; the bronchi contain muco-pus. The upper lobe is in the condition of gray hepatization; the quantity of fibrin is very large in the exudate. There are numerous areas of lung, one as large as a hen's egg, which are hepatized and necrotic, but not gangrenous. The right lower lobe is partly hepatized. The left upper lobe is hepatized and gray, the lower lobe is hepatized and red.

The blood vessels of the lung were artificially injected; the injection stopped short at the edges of the necrotic areas.

In rare cases there is an infiltration of the interlobular septa with pus.

CASE V. *Lobar Pneumonia; Infiltration of the Interlobular Septa with Pus.*—Male, 27 years old, December 25, 1891.

The man has been very intemperate and two years ago had a chancre.

Early this morning he began to have pain between the shoulders and over the upper part of the abdomen.

Yesterday he had been drinking heavily and was wet through with the rain. He vomited a great deal and began to cough and expectorate. The sputum was at first muco-purulent and frothy. The man is now well nourished, his tongue coated, pulse 100, breathing 24, temperature 105°.

Second day: Pulse 86-108, breathing 22-26, temperature 102.6°-104.2°; profuse muco-purulent and blood-stained sputum; urine 1020, albumin but no casts; great pain in the right side; diminished breathing over right lower lobe.

Third day: Pulse 108-120, breathing 24-28, temperature 104.6°-104.6°. He was cyanotic, delirious, and looked very badly. Over the right lower lobe there was dulness on percussion and bronchial voice and breathing.

Fourth day: Pulse 96-118, breathing 28-30, temperature 104.6°-105.6°. He continued to be cyanotic and actively delirious. He was treated with strychnine, aconitine, and digitaline without any effect.

Fifth day: Pulse 154, temperature 108°; he died.

Autopsy, seven hours after death.—The left lung is covered with old adhesions at the apex; the whole lung is congested; the bronchi are congested and coated with muco-pus.

The lower lobe of the right lung is coated with fibrin; the bronchi are full of pus; the whole lobe is in the condition of red hepatization; the interlobular septa are infiltrated with pus.

In the heart there is some thickening of the aortic and mitral valves.

In the liver there is commencing cirrhosis. The kidneys show the lesions of chronic nephritis, not far advanced.

The spleen is normal.

Very rarely after death we find thrombosis of the pulmonary arteries. This does not seem to change the ordinary course of the pneumonia during the life of the patient.

CASE VI. *Lobar Pneumonia; Thrombosis of the Pulmonary Arteries.*—Female, 33 years old, January 10, 1899.

Five days ago she began to have cough, soreness in the chest, aching all over the body, and felt sick.

Two days ago she went to bed with fever, pain in the left chest, vomiting, cough with muco-purulent sputum.

Now, on the sixth day, she is actively delirious. Over the left chest behind there is dulness on percussion, bronchial voice and breathing, and coarse râles.

She continued to be very ill with active delirium until the ninth day, when she died.

Autopsy.—The pleuræ of both lungs are coated with fibrin.

The lower lobe of the left lung is in the condition of red hepatization. There are firm thrombi in the pulmonary arteries of both lungs.

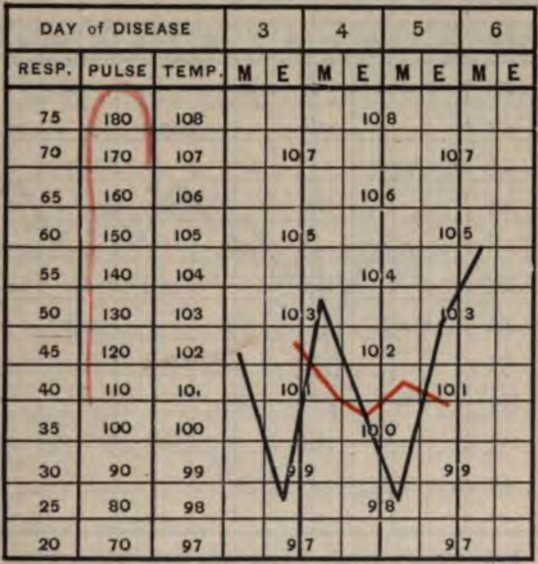
The heart is normal. The spleen is congested.

The liver is normal. The kidneys are atrophied and cystic.

(For Chart of Case VI, see Fig. 35.)

With every lobar pneumonia there is more or less inflammation of the larger bronchi. This is usually a catarrhal bronchitis, but sometimes the bronchi are filled with cords of fibrin.

FIG. 35



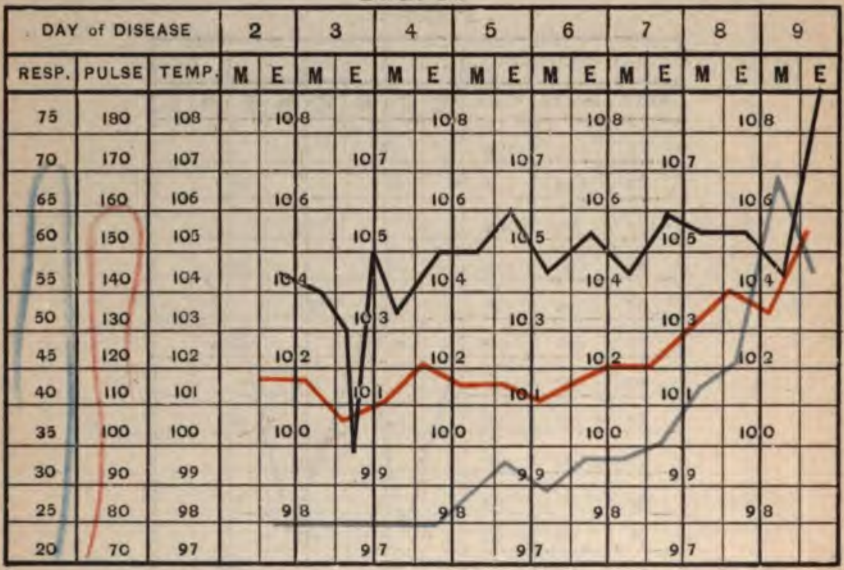
Temperature black, Pulse red.

CASE VI.—LOBAR PNEUMONIA: THROMBOSIS OF THE PULMONARY ARTERIES.

When the pneumonia is complicated with influenza severe forms of catarrhal bronchitis are often present. The patients cough up large quantities of mucous, purulent, and bloody serum. The dyspnœa is more troublesome, the temperature is higher, and the tendency to heart failure and venous congestion is more marked. Cases illustrative of this will be given with the description of influenza pneumonia.

Acute pericarditis is a serious complication. It may be that there will be nothing to call attention to the condition of the heart, and the case seems only to be a pneumonia of severe type. Or there may be decided precordial pain, a rapid and feeble pulse, a pericardial friction sound, rapid breathing, and cyanosis. It occasionally happens that the symptoms of the pericarditis are more marked than those of the pneumonia, so that it is even possible for the pneumonia to be overlooked.

FIG. 36



Temperature black, Pulse red, Respiration blue.

CASE VII.—LOBAR PNEUMONIA; PERICARDITIS.

CASE VII. *Lobar Pneumonia; Pericarditis.*—Male, 18 years old, March 26, 1896.

On the day before admission he began to have headache, cough with mucous sputum, fever, and pain on the right side of the chest.

Now there is sonorous breathing and coarse râles over both lungs.

On the third day he received two tub baths, which were followed by a temporary fall in the temperature.

On the fourth day there was dulness on percussion and bronchial voice and breathing over the lower lobe of the left lung.

On the sixth day he became actively delirious and the lungs were filling up with mucus. The delirium continued, the pulse became more and more rapid and feeble, and he died on the ninth day.

Autopsy.—The lower lobe of the left lung is entirely consolidated, the upper lobe is compressed. The whole right lung is intensely congested.

The pericardium is coated with fibrin and the pericardial sac is moderately distended with turbid yellow serum.

(For Chart of Case VII, see Fig. 36.)

Endocarditis with ulcerations and vegetations on the valves and septic symptoms due to a pneumococcus infection occurs by itself. It may also follow directly after a lobar pneumonia. The following case is an example:

CASE VIII. *Lobar Pneumonia; Prolonged Consolidation; Ulcerative Endocarditis.*—Male, 50 years old, June 14, 1903.

Two weeks ago, after exposure, a shaking chill, with pain in the right side, dyspnœa and prostration. He stayed in bed for three days and got up and walked around, but did not go back to work. The dyspnœa has increased, with cough, brownish sputum, and vomiting every day.

Now, over the right chest in front, below the third interspace, there is dulness and diminished breathing. Behind, below the angle of the scapula, there is flatness on percussion, bronchial voice and breathing, and crepitant râles.

On the twentieth day the man was improving; there was no pain in the chest, and no cough.

On the thirty-eighth day he had a chill, followed by rise of temperature, and his tongue became dry.

On the forty-third day, over the right lung behind, from the spine of the scapula to the base, there was dulness on percussion, bronchial voice and breathing, and diminished vocal fremitus.

On the fifty-fifth day the right chest was aspirated several times, but no fluid was found.

On the sixty-seventh day he died.

Autopsy.—The heart: There are small fresh vegetations just above the tricuspid valve. Just above the attached border of the mitral valve is an ulcerated surface. The aörtic valve is ulcerated and covered with vegetations.

The lower lobe of the right lung is in the condition of smooth, mottled consolidation. The lower lobe of the left lung is congested and œdematous.

The stomach and intestines are normal. The liver is fatty. The kidneys are in a condition of acute degeneration.

(For Chart of Case VIII, see Fig. 37.)

Perhaps the rarest complication of lobar pneumonia is thrombosis of the coronary arteries of the heart, causing death after defervescence and resolution.

CASE IX. *Lobar Pneumonia; Thrombosis of the Coronary Arteries; Sudden Death.*—A man, 28 years old, went through a lobar pneumonia and defervesced on the seventh day. Seven days after convalescence had fairly set in, he suddenly fainted, became pulseless, and in fifteen minutes was dead.

Autopsy.—There is a thrombus, or embolus, in the left middle cerebral artery. Both lungs are congested and œdematous. In both coronary arteries are firm, white, dry thrombi. One of these thrombi increases to the size of a chestnut and lies floating above the aortic valves in the aorta. Another firm, white thrombus, the size of a chestnut, is impacted behind one of the aortic valves. The heart cavities contain a little fluid blood.

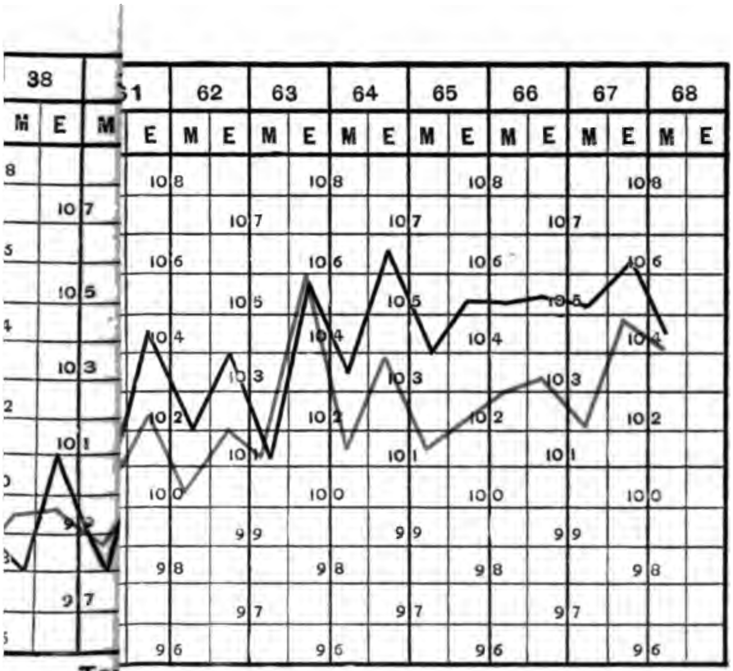
Acute meningitis occurs as a complicating inflammation, due to the same infection as the pneumonia, in a moderate number of cases. The patients regularly die. The meningitis may run its course without any distinctive symptoms, or the delirium may be more active with the contractions of groups of muscles, general convulsions or paralyses.

CASE X. *Lobar Pneumonia; Meningitis.*—Female, 48 years old, December 26, 1896.

About two weeks ago she began to have a dry cough, pain in the left side, vomiting, and diarrhœa. Four days ago she had a chill, followed by fever and prostration.

Yesterday she became much worse, was unconscious and apparently paralyzed on the left side.

Now the temperature is 105°, pulse 156, breathing 44; urine 1028, no albumin. She is unconscious; the left side is paralyzed, with muscular twitchings; the pupils are dilated. Over the upper part of the right lung there was dulness on percussion, bronchial voice and breathing, and subcrepitant râles. She remained in the same condition until she died two days later.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VIII.—Lo

Autopsy.—The pia mater is infiltrated with fibrin and pus.

The upper lobe of the right lung is in the condition of red hepatization.

There is old thickening of the aortic and mitral valves, with hypertrophy of the left ventricle.

The kidneys show the lesions of a chronic nephritis without exudation.

CASE XI. *Lobar Pneumonia; Meningitis.*—Male, 25 years old, January, 1881.

Four days before death he had a chill.

Two days before death he had headache, delirium, and involuntary contractions of the muscles of the face and arms.

Autopsy.—The pia mater is congested and infiltrated with pus cells.

The lungs: The right pleura is coated with fibrin and there is a considerable amount of purulent serum in the pleural cavity. The middle lobe of the right lung is in a condition of red hepatization.

Acute peritonitis, as a complication apparently due to a pneumococcus infection, is of very rare occurrence.

CASE XII. *Lobar Pneumonia; Peritonitis.*—Female, 37 years old, March 2, 1880.

Ten days ago she had a chill, pain in the right chest, dyspnœa, fever, cough, and expectoration.

Now, on the eleventh day, she looks very sick, with labored breathing, cough, and thick muco-purulent sputum. The temperature is 103°, pulse 128, breathing 44. There is dulness and bronchial breathing over the upper lobe of the right lung.

On the twelfth day she died.

Autopsy.—The heart is normal. The upper lobe of the right lung is in a condition of gray hepatization.

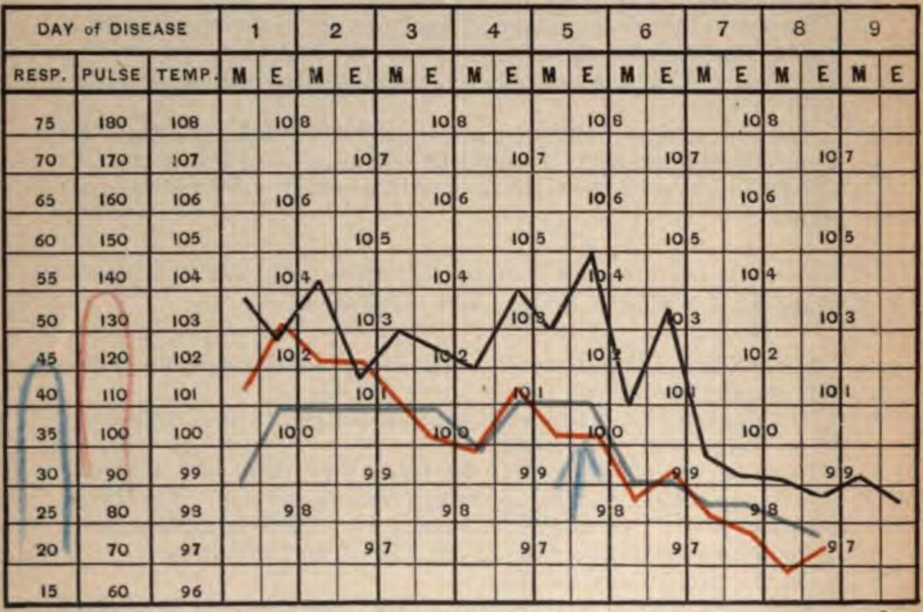
The pulmonary pleura of the left lung is coated with fibrin and pus.

The peritoneum is coated with fibrin and pus.

A purulent inflammation of the large joints, due to pneumococcus infection, has been described by a number of observers. The knee joint is the one usually affected, but it may also involve the shoulder. In the cases that I have seen, the symptoms of septic infection have been more marked than the local symptoms of the synovitis. In fact, the inflamed joint may be overlooked altogether and the high temperatures attributed to the pneumonia.

The Course of the Disease.—1. The regular cases. These may be mild or severe, they may terminate in recovery or in death, but they all have this in common, that the clinical picture is that of an inflammation of the lung with only moderate evidence of general poisoning. The patients begin with the chills, rapid rise of temperature, sleeplessness and restlessness, vomiting, pain in the side, cough, expectoration, and dyspnœa. These symptoms continue mildly or severely; after the third day comes the liability to heart failure, and finally, at about the seventh day of the disease, comes defervescence or death. The severity of the symptoms is in proportion to the extent of lung involved and to the intensity of the inflammation.

FIG. 38



Temperature black, Pulse red, Respiration blue.

CASE XIII.—LOBAR PNEUMONIA, REGULAR CASE.

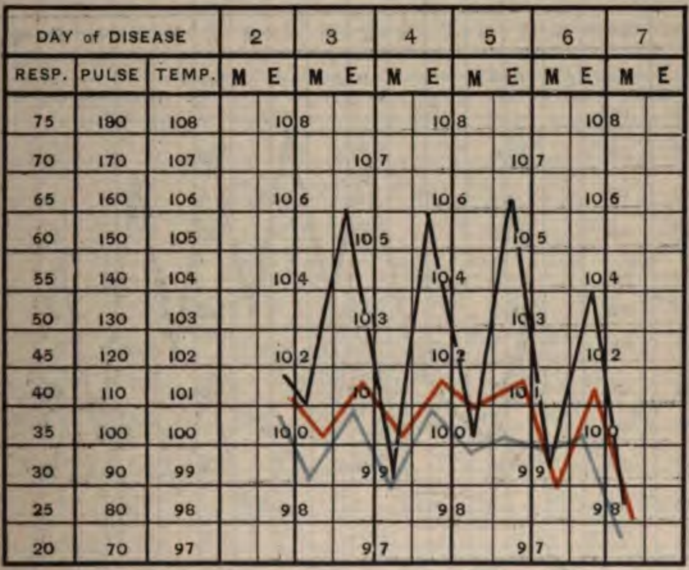
CASE XIII. *Lobar Pneumonia, Regular Case; Recovery.*—Male, 18 years old, December 30, 1896.

This morning he woke up feeling feverish, with headache, vomiting, and general pains.

Now his tongue is red and dry. Over the lower lobe of the right lung there is dulness on percussion and bronchial voice and breathing. He continued to be fairly sick until the seventh day of the disease, when he defervesced. Convalescence was rapid, and six days later he was discharged cured.

(For Chart of Case XIII, see Fig. 38.)

FIG. 39



Temperature black, Pulse red, Respiration blue.

CASE XIV.—LOBAR PNEUMONIA, IRREGULAR TEMPERATURE.

This ordinary picture of the disease may be varied by an unusual difference between the morning and evening temperatures.

CASE XIV.—The patient, an adult, had a regular lobar pneumonia involving the lower lobe of the right lung, which ran its ordinary course, without the use of drugs, and terminated in recovery.

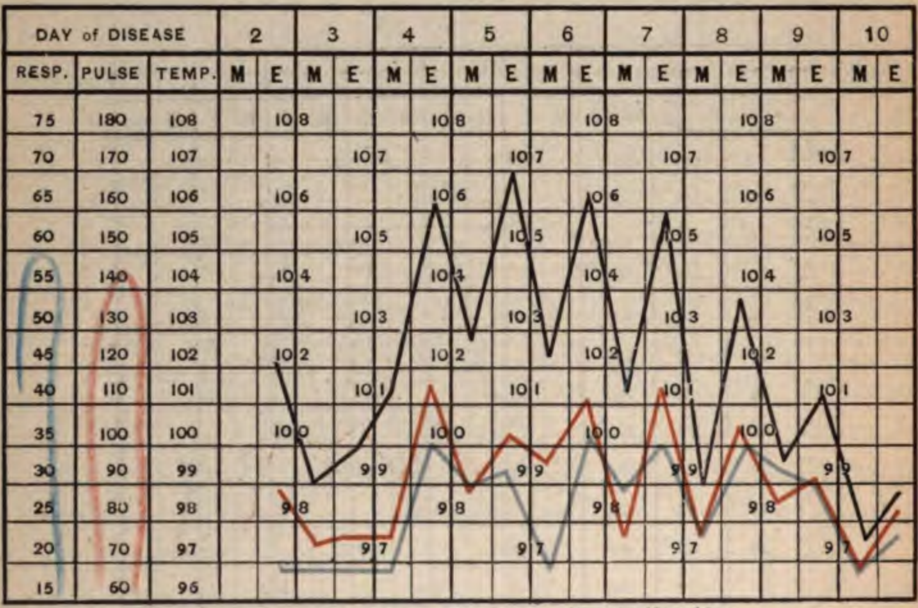
(For Chart of Case XIV, see Fig. 39.)

There are also cases with unusually high temperatures, but otherwise a normal course.

Case XV.—The patient, an adult, had a pneumonia which involved the lower lobe of the left lung and the upper lobe of the right lung. He was seriously ill, but defervesced on the ninth day and recovered. No antipyretics or quinine were used.

(For Chart of Case XV, see Fig. 40.)

FIG. 40



Temperature black, Pulse red, Respiration blue.

Case XV.—Lobar Pneumonia, high temperatures.

Curious falls of temperature can be produced by cold tubbing, or by packing the body in ice. The temperature falls quickly several degrees and then rapidly rises. No particular effect is exerted on the whole course or duration of the disease.

Case XVI. *Lobar Pneumonia; Treatment by Cold Baths.*—Female, 9 years old, June 10, 1900.

Three days ago, after exposure to cold, she began to have cough with muco-purulent sputum, headache, vomiting, diarrhœa, and fever.

Now, on the fourth day, she is dull and stupid, with a good deal of pain in the head and chest. Over the upper lobe of the right lung there is dulness on percussion, bronchial voice and breathing, and subcrepitant râles.

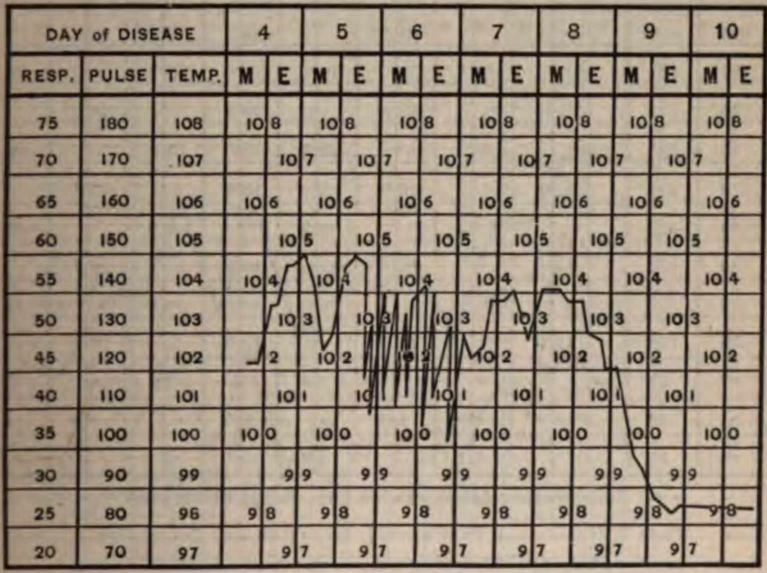
On the fifth and sixth days she was given tub baths, with the effects on the temperature shown in the chart.

On the ninth night she defervesced.

On the fourteenth day she was discharged cured.

(For Chart of Case XVI, see Fig. 41.)

FIG. 41



Temperature black.

CASE XVI.—LOBAR PNEUMONIA; BATHS.

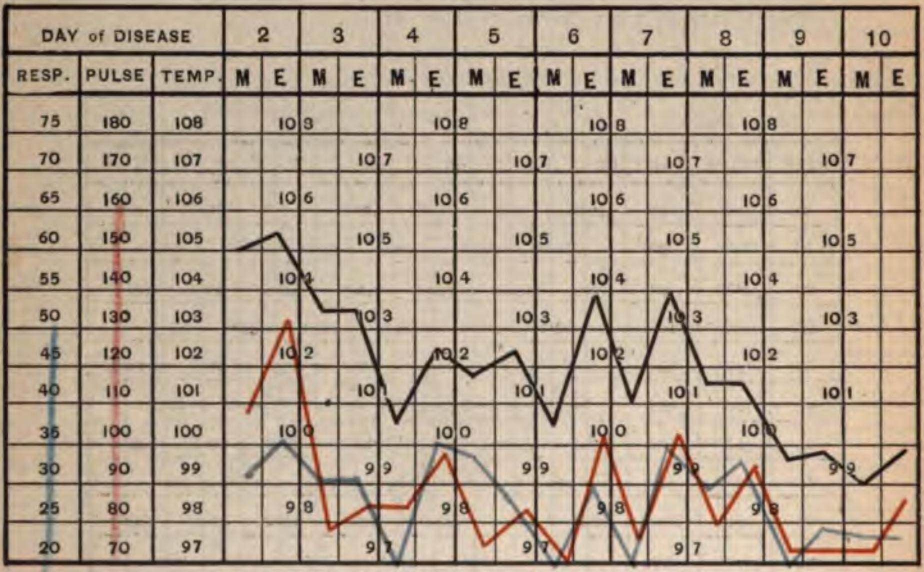
In a certain number of cases, while the temperature continues fairly high, it is possible to keep the pulse below 100 by the use of small doses of aconitine and digitaline. The patient's general condition seems to be materially improved by this.

CASE XVII.—The patient had a pneumonia of the right lower lobe; he defervesced on the ninth day and recovered. The temperature ran its ordinary course, but the pulse was kept at or below 100 by the use of aconitine and digitaline.

(For Chart of Case XVII, see Fig. 42.)

2. There are cases which run a short course of moderate severity, with very little evidence of infection, and defervesce on the third or fourth day. They do this either with or without treatment.

FIG. 42



Temperature black, Pulse red, Respiration blue.

CASE XVII.—LOBAR PNEUMONIA; ACONITINE AND DIGITALINE.

CASE XVIII. *Lobar Pneumonia; Defervescence on the Third Day.*—Male, 24 years old, December 11, 1895.

On the day of admission he had a shaking chill, followed by fever and pains all over the body. There was no cough or dyspnœa.

Now over the left chest behind there is a little dulness on percussion, harsh breathing, with crepitant and subcrepitant râles. He continued to be moderately sick, without cough, until the third day of his illness, when he defervesced.

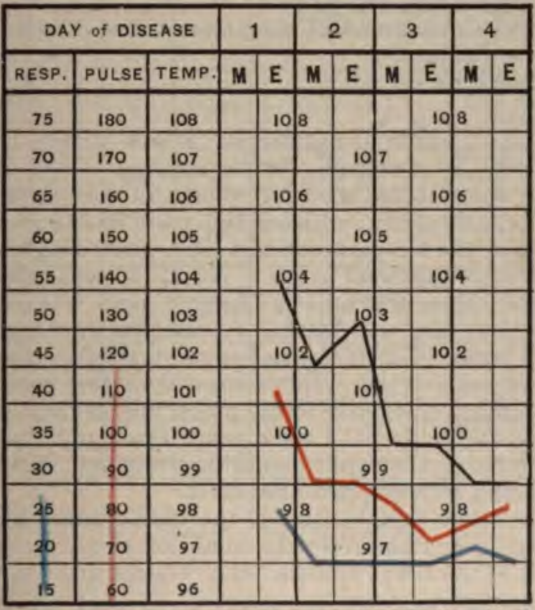
On the fourth day the physical signs had disappeared.

(For Chart of Case XVIII, see Fig. 43.)

3. There are cases which last only for a few hours and terminate fatally before the lung is consolidated. This belongs especially to old people.

CASE XIX. *Lobar Pneumonia; Death at the End of Four and a Half Hours.*—A woman, 70 years old, had been in the hospital for a year suffering from a number of trifling ailments. On November 29, 1880, after feeling chilly during the night, at half-past 6 in the morning she was suddenly seized with rapid breathing, feeble pulse, and great prostration. At half-past nine the temperature was 100°, pulse 132, breathing 48. At 11 o'clock she died.

FIG. 43



Temperature black, Pulse red, Respiration blue.

CASE XVIII.—LOBAR PNEUMONIA, THREE DAYS.

Autopsy.—The larynx, trachea, and large bronchi are congested and coated with mucus.

The pericardium is congested and there is a little turbid serum in the pericardial sac.

There is no fibrin on the pulmonary pleura. Both lungs are congested and œdematous.

The upper lobe of the right lung is in the condition of commencing red hepatization. The air spaces of this lobe contain granular matter, a little reticulated fibrin, a few pus cells and a few epithelial cells. The epithelial cells on the walls of the air vesicles are increased in size and number. The capillaries are filled with white blood cells.

The liver is large, hard, and smooth. The gall bladder contains a large calculus. The large bile ducts in the liver are dilated and contain calculi.

The spleen is large and firm.

The mucous membrane of the stomach and small intestine is coated with mucus.

The kidneys are normal.

CASE XX. *Lobar Pneumonia; Death after Twenty-four Hours.*—Male, 80 years old. After retiring to bed at 11 in the evening he was attacked with severe pain in the epigastric region. At 7 o'clock the next morning the pain had shifted more to the right side, the temperature was 101°, pulse 100, breathing 30. There were no physical signs. At 12 o'clock the pain had disappeared; there were still no physical signs. At 5 o'clock that afternoon there was well-marked crepitation over the right middle lobe in front. The face was becoming anxious, the pulse was feebler and more rapid. He continued in this condition, with rapid breathing, quick and feeble pulse, the feet and hands gradually becoming colder, the intelligence clear, until at 11 o'clock the same evening the rapid breathing grew first slower, then interrupted, and then stopped altogether.

Autopsy.—The middle lobe of the right lung and the upper part of the lower lobe are in the condition of red hepatization.

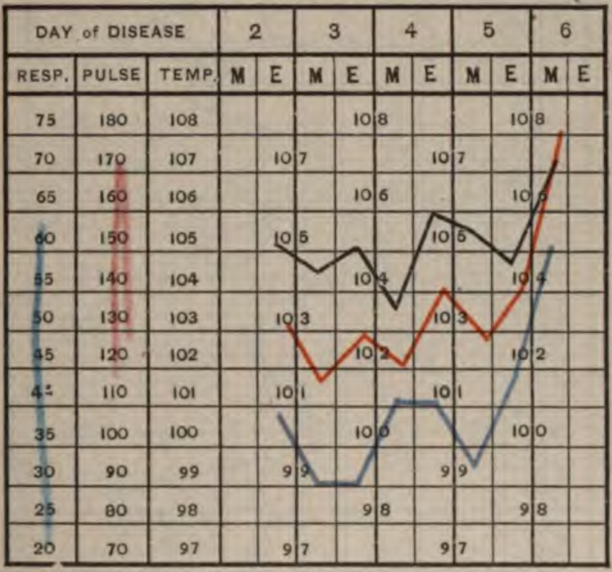
The rest of the lungs and the other viscera are normal.

4. There are cases in which a considerable portion of both lungs is inflamed. In some of these cases a large portion of both lungs is involved at once, in some first one lobe and then others are involved, in some there is so much congestion and œdema of the unhepatized portions of lung that they are rendered useless for respiration. These cases usually die before the sixth day of the disease.

CASE XXI.—A man, 21 years old, was kicked overboard from a canalboat into the river. On the second day after this accident he was attacked with a chill, dyspnœa, and vomiting. Two days after this he died.

Autopsy.—The entire right lung is in the condition of red and gray hepatization, and there are numerous patches of red hepatization in the left lung.

FIG. 44



Temperature black, Pulse red, Respiration blue.

CASE XXIII.—LOBAR PNEUMONIA, BOTH LUNGS.

CASE XXII.—Female, 29 years old, February 23, 1875.

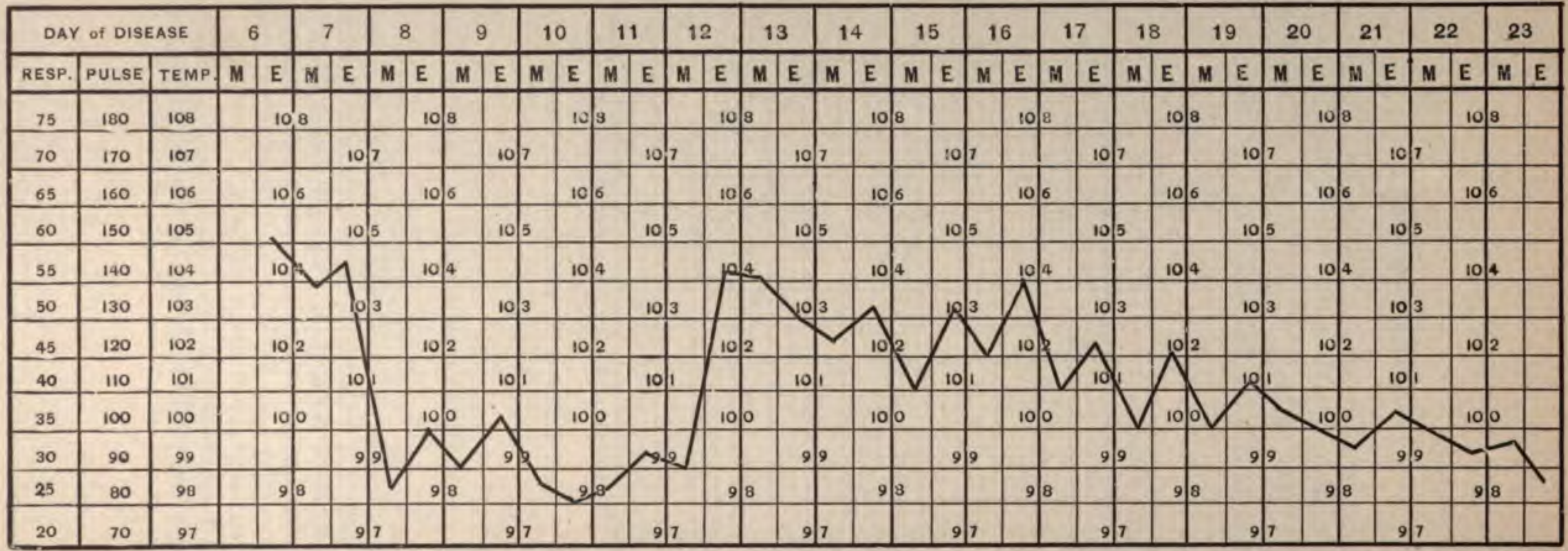
She came into the hospital without any history and died the same day.

Autopsy.—Both lungs are intensely congested and œdematous, with commencing red hepatization of all the lobes of both lungs. There is a little fibrin on the pulmonary pleura. There are no changes in the bronchi. The air spaces are not dilated. The inflammation is diffuse over both lungs. Some of the air spaces are empty, some show an increase of the epithelial cells, some contain reticulated fibrin and pus.

The heart is normal.

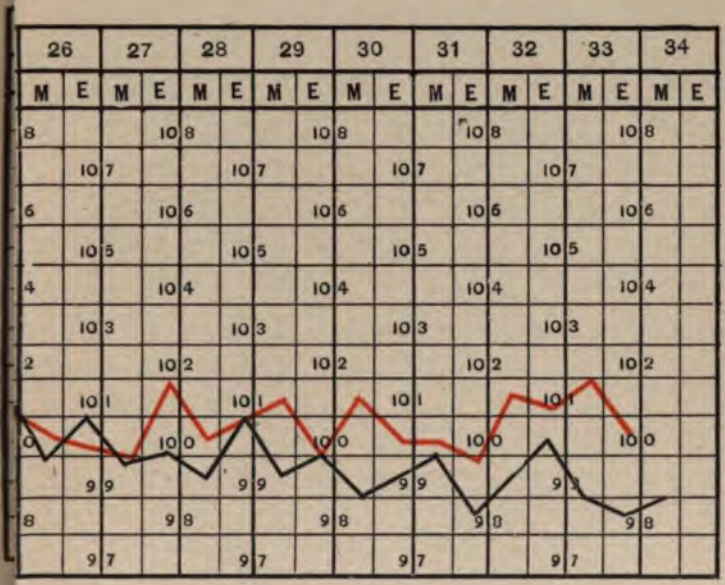
The kidneys show the lesions of chronic nephritis.

FIG. 15



Temperature black.

Case XXIV.—Lobar Pneumonia; Recurrence.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CASE XXIII. *Lobar Pneumonia involving Both Lungs.*—Female, 30 years old, January 1, 1895.

The day before admission to the hospital she began to have chills, fever, headache, vomiting, and cough.

Now there is dulness over the upper part of the right lung in front and behind. She became steadily worse, was delirious, and died on the sixth day.

Autopsy.—The left lung is œdematous, with irregular patches of red hepatization; there is no fibrin on the pleura; the bronchi contain muco-pus. The upper lobe of the right lung is in the condition of gray hepatization, the lower lobe is in the condition of incomplete red hepatization.

The heart is normal.

The kidneys show the lesions of chronic nephritis.

(For Chart of Case XXIII, see Fig. 44.)

5. It sometimes happens that a patient has a pneumonia which runs its course and terminates in recovery. Then, after an interval of several days, there is a fresh attack of pneumonia in the other lung or the same lung.

CASE XXIV. *Pneumonia of the Right Lung; Recovery followed by Pneumonia of the Left Lung.*—Female, 24 years old, December 1, 1898.

Five days ago, after exposure to cold and wet, she had a chill, fever, prostration, cough, and pain in the right side.

Now the breathing is rapid, shallow, and painful; she looks very sick. Over the upper lobe of the right lung behind there is dulness on percussion, with bronchial voice and breathing.

On the eighth day of the disease she defervesced.

On the eleventh day she was out of bed.

On the twelfth day the temperature ran up and she was again sick.

On the fourteenth day there was dulness, with bronchial voice and breathing, over the lower lobe of the left lung.

On the eighteenth day she began to improve, and by the twenty-third day the temperature fell to normal.

(For Chart of Case XXIV, see Fig. 45.)

CASE XXV. *Lobar Pneumonia; Recurrence after Nine Days in the same Lung.*—Male, 42 years old, November 25, 1885.

For two months past he has not been in his usual health; he has had cough with muco-purulent sputum, sometimes streaked with blood, and has lost appetite, flesh, and strength, but continued to work.

Four days ago he had a well-marked chill, followed by vomiting, dyspnœa, pain in the right side, and cough with blood-stained sputum.

Now, on the fifth day, he is fairly nourished, his tongue is moist and coated, there is cough with rusty sputum, the breathing is rapid and shallow; urine 1021, albumin. There are coarse râles over both lungs. Over the right lower lobe there is dulness on percussion, bronchial breathing, and subcrepitant râles.

On the ninth day he had great pain in the right side, the breathing was hurried and insufficient, the tongue was dry and brown.

On the twelfth day, over the entire right lung behind and in front there was dulness on percussion. Over the right upper lobe there was bronchial voice and breathing; over the lower lobe there was diminished breathing and coarse râles.

On the fourteenth day he defervesced.

On the seventeenth day he continued to improve. All the physical signs had disappeared, except a little dulness and a few subcrepitant râles over the right lung.

On the twenty-first day he was out of bed and eating solid food.

On the twenty-third day, during the night, he felt chilly and was suddenly seized with intense dyspnœa. The pulse, breathing, and temperature ran up. Over the right lower lobe there was dulness on percussion and bronchial voice and breathing.

On the twenty-fourth day he was cyanotic, with bad breathing, cough, delirium, and pain in the right chest.

On the twenty-fifth day he seemed to be rather better.

On the twenty-sixth day he was worse, with violent paroxysms of coughing, and a dry, brown tongue.

Between the twenty-seventh and twenty-eighth days he defervesced for the second time.

On the thirty-fifth day there was still dulness and increased voice and fremitus over the right lower lobe. A needle in the chest gave no fluid.

On the thirty-ninth day he was out of bed. He gained strength slowly and did not leave the hospital until the sixty-sixth day.

(For Chart of Case XXV, see Fig. 46.)

6. There are cases in which the evidences of the pneumococcus infection are very marked and out of proportion to the extent of inflammation in the lung.

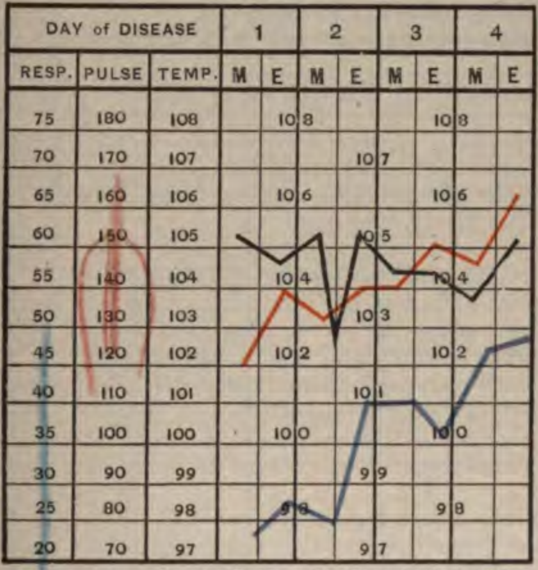
Case XXVI.—A woman, 32 years old, had suffered from chronic bronchitis for five months. Twelve days before her death, after exposure to cold and wet, she was seized with chills followed by fever.

On the second day of the pneumonia she had pain in the left side and dyspnœa.

On the fifth day she began to vomit, and after that vomited everything she ate.

On the eighth day she was admitted to the hospital; pulse 122, temperature 106° F., breathing 42; the tongue was dry and brown; there was great dyspnœa, and pain in the side.

FIG. 47



Temperature black, Pulse red, Respiration blue.

CASE XXVII.—LOBAR PNEUMONIA, SEPTIC CASE.

On the ninth day, pulse 107, temperature 103° F., breathing 35.

On the eleventh day, pulse 160, temperature 105° F., breathing 48.

On the twelfth day she died.

Autopsy.—Only the upper lobe of the left lung is hepatized, while the rest of the lungs shows the lesions of emphysema and chronic bronchitis.

CASE XXVII. *Lobar Pneumonia; General Infection.*—Female, 16 years old.

February 1, 1902, she had an attack of measles, which ran a regular course.

February 8 she began to look worse, the white blood cells numbered 20,000, but there was no change in the breathing and no physical signs.

On the second day of the pneumonia she looked worse; the breathing was rapid and insufficient; there was dulness and diminished breathing over the left lower lobe.

On the third day there was marked mucous rattling all over both lungs; the breathing was very bad; the dulness and diminished breathing could not be made out.

The breathing continued to be very bad until she died on the fourth day.

(For Chart of Case XXVII, see Fig. 47.)

CASE XXVIII. *Lobar Pneumonia, Septic Form, without Physical Signs.*—Male, 55 years old, December 13, 1875.

Two months ago he came to New York from China and has been drinking freely ever since.

Four days ago he was suddenly seized with a chill, vomiting, headache, cough, and pain in the right chest.

Now, on the fifth day, the tongue is coated and moist, temperature 100° F., pulse 92; he is breathing quietly; there are no physical signs; he complains chiefly of headache.

On the sixth day, temperature 103°-104° F., pulse 88-96.

On the seventh day, temperature 103°-104° F., pulse 100-104.

On the eighth day, temperature 102°-104° F., pulse 100-104. He was gradually becoming worse, with a dry tongue and increasing apathy.

On the ninth day, temperature 105°-105° F., pulse 120; he was fairly in the typhoid state, with rapid and labored breathing.

I examined the chest every day and found no physical signs. The resonance and breathing over both lungs were always imperfect.

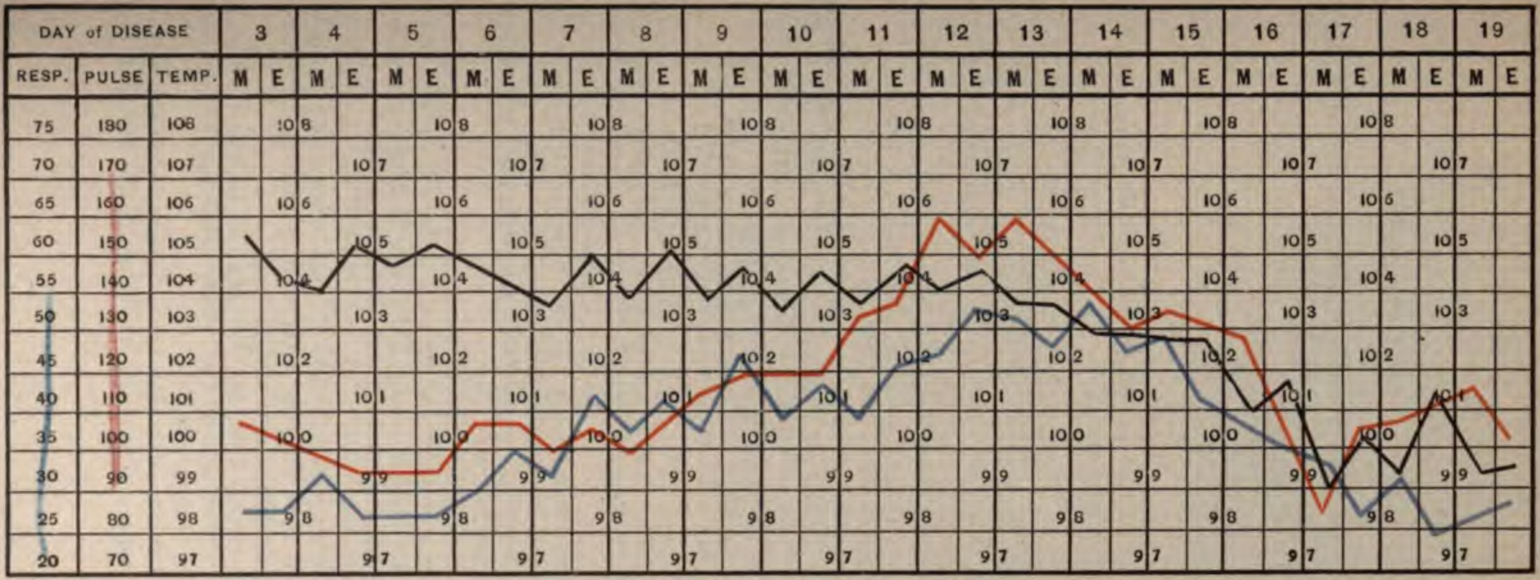
On the tenth day he died.

Autopsy.—The brain was not examined. The heart is normal, except for a little old thickening of the aortic and mitral valves.

There are old pleuritic adhesions over the whole of the right lung.

Over the middle lobe the old adhesions are infiltrated with pus. The middle lobe and the adjacent portions of the upper and lower lobes are in the condition of red and gray hepatization. The corresponding bronchi, except the largest, are filled with cords of fibrin.

FIG. 48



Temperature black, Pulse red, Respiration blue.

Case XXIX.—Lobar Pneumonia, protracted.

The left lung is covered with old pleuritic adhesions; there is no bronchitis, no congestion, no œdema.

The liver is congested.

The spleen is large and firm.

The stomach and intestines are normal.

The kidneys are congested.

7. There are cases which continue for an unusual length of time, but yet may recover.

CASE XXIX. *Lobar Pneümonia, Protracted.*—Male, 18 years old, March 28, 1895.

Two days ago he had a chill, vomiting, cough with rusty sputum, and pain in the right side.

Now, on the third day, over the right lower lobe there is dulness on percussion, bronchial voice and breathing.

On the fifth day he was delirious.

He continued in the same condition, delirious, with a brown, dry tongue, until the fifteenth day, when he began to improve.

On the sixteenth day his temperature fell to the normal. He was treated with aconitine and digitaline.

On the thirtieth day he was discharged cured.

(For Chart of Case XXIX, see Fig. 48.)

CASE XXX. *Lobar Pneumonia, Prolonged Case; Thrombosis of the Left Femoral Vein.*—Male, 17 years old, February 16, 1897.

For two weeks he has not felt well and has had a cough, but continued at work until four days ago. Then he had a chill, followed by fever, pain in the left side, dyspnœa, and cough; he also vomited and felt very ill.

Now, on the fifth day, there is dulness on percussion and bronchial voice and breathing over the lower lobe of the left lung.

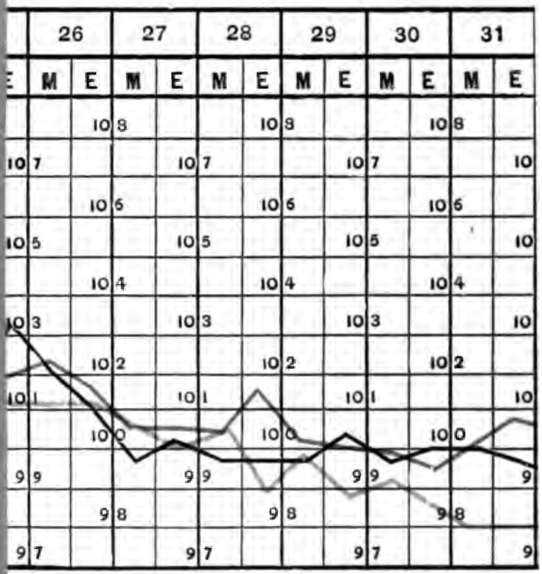
The man looks seriously ill, with a flushed face, coated tongue, and dyspnœa; urine 1022, no albumin or casts.

The man continued to be very ill until the sixteenth day, when the temperature fell to 101° F. and he seemed to be better.

But the next day the temperature rose again; the man was cyanotic, delirious, and looked badly.

On the twenty-first day he began to improve. This improvement continued, and by the twenty-ninth day he was much better and eating chopped beef.

On the thirty-sixth day he developed a thrombosis of the left femoral vein, followed by swelling of the leg and thigh. The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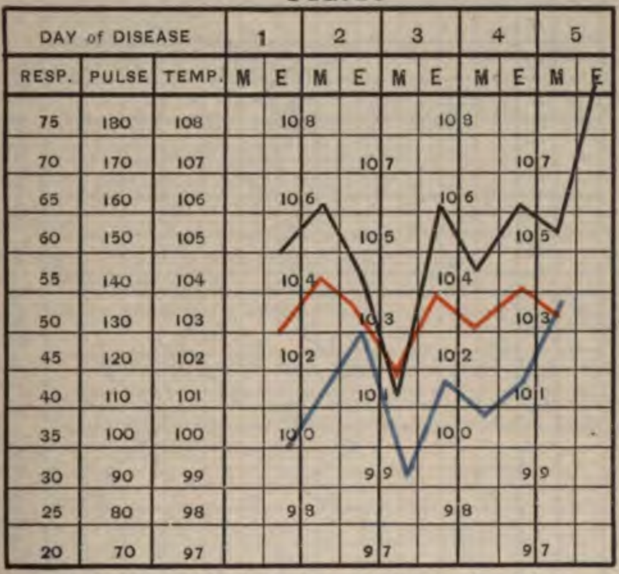
swelling of the leg continued, but otherwise the convalescence was uninterrupted until he left the hospital on the sixty-eighth day.

(For Chart of Case XXX, see Fig. 49.)

8. There are cases which are complicated by alcoholic poisoning. Usually the patients are drinking hard at the time they are attacked with the pneumonia. They are regularly very sick, with high temperatures, active delirium, or delirium tremens.

FIG. 50



Temperature black, Pulse red, Respiration blue.

CASE XXXI.—LOBAR PNEUMONIA: DELIRIUM TREMENS.

CASE XXXI. *Lobar Pneumonia; Delirium Tremens.*—Male, 24 years old, April 15, 1896.

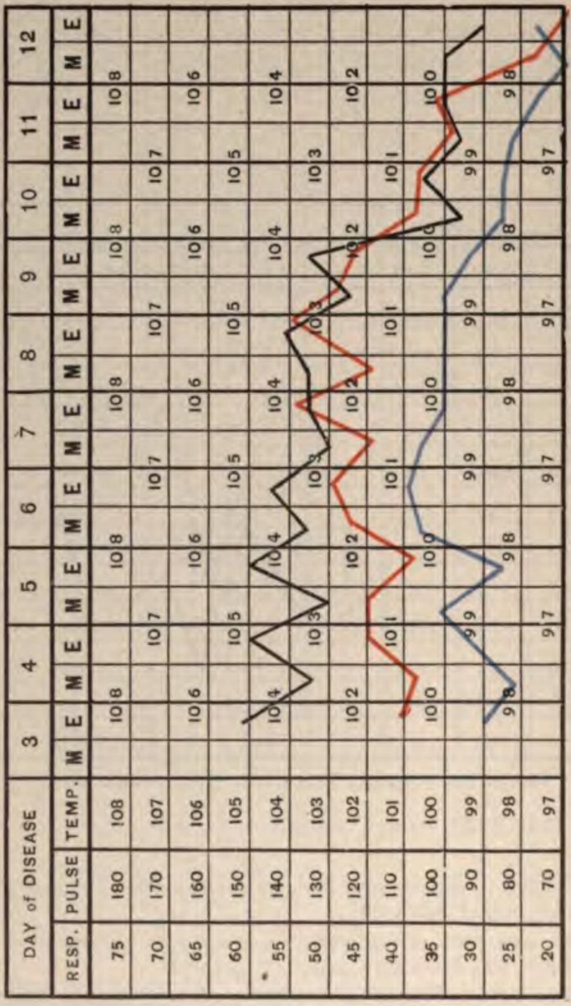
The man has been intemperate for several years, and for the past few days has been drinking very hard.

This morning at 6 o'clock he had a severe chill, followed by fever, pain in the chest, headache, cough and mucous sputum. Now over the lower lobe of the left lung there is dulness on percussion and diminished voice and breathing; over the rest of the lung there are coarse râles.

On the fourth day he began to have violent delirium with delusions and refused all medicine. He continued in this condition until he died on the fifth day.

(For Chart of Case XXXI, see Fig. 50.)

FIG. 51



Temperature black, Pulse red, Respiration blue.

CASE XXXII.—LOBAR PNEUMONIA; DEFERVESCENCE DELIRIUM.

9. Delirium at the time of defervescence. I have already called attention to the importance of distinguishing this form of delirium from alcoholic delirium and the ordinary delirium of

the disease. It comes on at the time of defervescence; it may or may not be accompanied by a rise of temperature; it lasts for days or weeks; the patients usually recover, but old persons sometimes remain permanently insane.

CASE XXXII. *Lobar Pneumonia; Delirium after Defervescence.*—Male, 28 years old, October 24, 1895.

Two days before admission he had a chill, cough with rusty sputum, pain in the right chest, and dyspnœa.

Now, on the third day, there are coarse râles over both lungs; over the right lower lobe there are bronchial voice and breathing and crepitant râles.

The man had a good deal of bronchitis, but did well.

On the ninth day he defervesced.

On the tenth day he began to be sleepless and delirious. The delirium continued until the fifteenth day. After that he did well and was discharged on the eighteenth day.

(For Chart of Case XXXII, see Fig. 51.)

CASE XXXIII. *Lobar Pneumonia; Delirium with Defervescence.*—Male, 19 years old, February 8, 1897.

On the afternoon of the day before admission he began to have severe pains in the right side, vomiting, cough, chilliness, and dyspnœa.

Now, on the second day, the face is flushed, the tongue is coated and dry; urine 1016, no albumin. Over the lower lobe of the right lung there is dulness on percussion, diminished breathing, and subcrepitant râles.

The man slept very little, and on the fifth day developed a noisy delirium.

On the sixth day he defervesced. He continued to be delirious, with a little fever, until the twelfth day. After that time he improved and left the hospital entirely well on the twenty-fifth day.

(For Chart of Case XXXIII, see Fig. 52.)

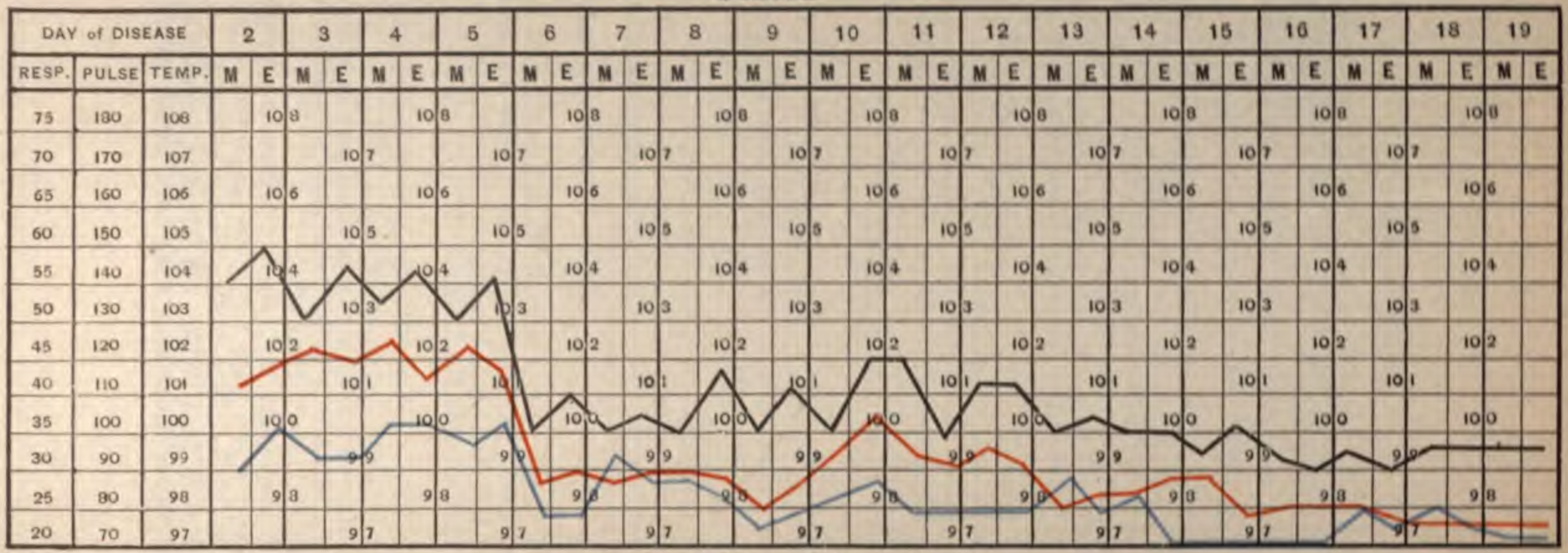
CASE XXXIV. *Lobar Pneumonia; Delirium at time of Defervescence.*—Male, 27 years old, March 3, 1897.

On the afternoon of the day before admission he began to have severe headache, pain in the left side, dyspnœa, chill, fever, and cough with blood-stained sputum.

Now, on the second day, the prostration and dyspnœa are marked; over the entire left lung there is dulness and diminished voice and breathing; urine 1020, a trace of albumin.

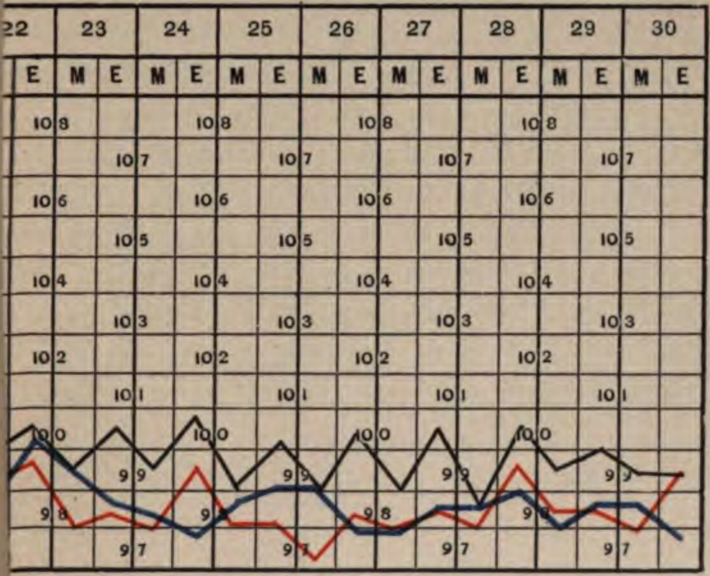
The man went on with high but irregular temperatures untii the seventh day of the disease, when there was an attempt at de-

FIG. 52



Temperature black, Pulse red, Respiration blue.

CASE XXXIII.—LOBAR PNEUMONIA; DEFERVESCENCE DELIRIUM.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fervescence. After this his temperature ran from 99° to 102° until the twenty-eighth day, when it reached the normal. During this time the man's general condition steadily improved.

On the sixth day, the day before defervescence, the man became actively delirious. This delirium continued, although much less active, and was still present when he left the hospital on the thirty-first day.

(For Chart of Case XXXIV, see Fig. 53.)

10. General convulsions in the course of lobar pneumonia are of rather rare occurrence; I have seen five cases.

In four of these cases there was present chronic nephritis of long standing. In such cases the convulsions seemed to be due to an exacerbation of the nephritis and a more acute development of renal symptoms.

In one case, however, the kidneys were only in the condition of acute degeneration.

In one case the convulsions occurred on the second day of the disease, in the others not until near its termination.

In one case the pneumonia was double, in two cases it involved an entire lung, in two cases only one lobe.

The brain was in one case normal, in two cases it showed an increase of serum beneath the pia mater, in two cases it was not examined.

11. There are cases in which a pneumonia follows so soon after the inhalation of chloroform, ether, or smoke that it seems to be due to these irritants. Apparently in these persons the pneumococcus infection would not have taken place if the lungs had not first been irritated.

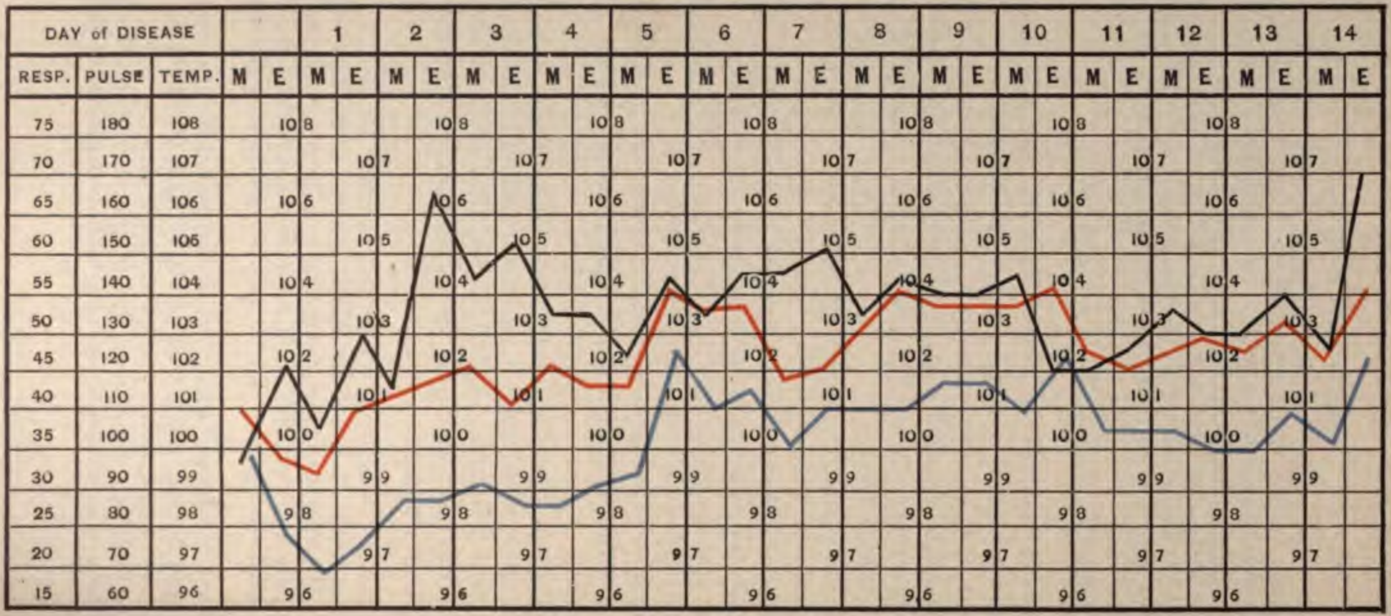
Case XXXV.—Male, 46 years old, January 10, 1900.

This morning at a fire he inhaled large quantities of dense smoke. He became unconscious and remained so for an hour. He was pale and cyanotic, with a feeble pulse; breathing embarrassed and stridulous. There were no physical signs over the lungs.

The next day he was somewhat better, but still very weak. In the evening he was taken with chilly sensations, sweating, prostration, weak pulse, and severe pain in the chest.

On the next day, the second day of the pneumonia, he had no voice; there was stridulous cough with profuse muco-purulent sputum.

FIG. 54



Temperature black, Pulse red, Respiration blue.

Case XXXV.—Lobar Pneumonia from Smoke.

On the third day of the pneumonia the laryngitis was better; the pulse was very weak; the sputum was now rusty and contained pneumococci.

On the fourth day the pulse and breathing were better. There was dulness on percussion, with bronchial voice and breathing over the lower lobe of the right lung.

On the fifth day he was delirious.

He continued to be delirious and very ill, and on the tenth day the physical signs of consolidation of the lower lobe of the left lung were present.

On the thirteenth day, hæmoglobin 85 per cent, red cells 5,544,000, white cells 23,300.

On the fourteenth day he died.

This man contracted his pneumococcus infection in the hospital ward.

(For Chart of Case XXXV, see Fig. 54.)

12. The pneumonia of old persons often runs an irregular course. The extent of lung inflamed may be small and the physical signs uncertain—a little dulness on percussion, a few subcrepitant râles, a diminished intensity of breathing—or even no physical signs at all. The patients usually have chilliness, or a distinct chill, to mark the invasion of the disease, and more or less fever while it is running its course. The appetite is lost, and there may be nausea and vomiting. The pulse is rapid and often feeble. Either stupor or a mild delirium is often present. The prostration is out of proportion to the extent of lung inflamed. The characteristic cough, expectoration, and pain in the chest are absent altogether, or imperfectly developed; even the breathing may not be at all changed. But the disease is very fatal in old persons, and some of them die quite suddenly after seeming to be only moderately sick for a few days.

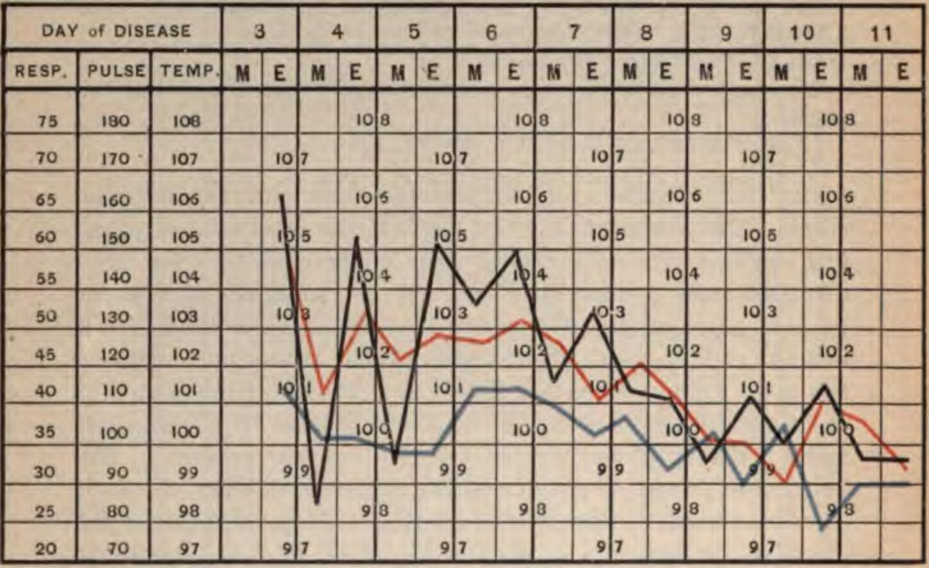
13. At the present time in New York a large number of lobar pneumonias occur in the course of an influenza. There is a double infection with the influenza bacillus and the pneumococcus. This does not produce broncho-pneumonia, but lobar pneumonia combined with severe catarrhal bronchitis. In some cases the inflamed lung is hepatized in the usual way, but in others with a good deal of congestion there is comparatively little exudation. The combinations of incomplete consolidation with bronchitis give irregular physical signs. In a considerable number of

cases the disease terminates by lysis rather than by crisis. Protracted cases are not uncommon.

The influenza may precede the pneumonia for several days, both may come on at the same time, or the pneumonia may run its course and resolve, but the influenza still continue.

It is important in these patients to try to distinguish how much of the illness is due to influenza and how much to pneumonia.

FIG. 55



Temperature black, Pulse red, Respiration blue.

CASE XXXVI.—LOBAR PNEUMONIA; INFLUENZA; NEPHRITIS.

CASE XXXVI. *Lobar Pneumonia; Influenza; Exudative Nephritis.*—Female, 22 years old, March 21, 1891.

Two weeks ago she began to have chills, fever, nausea and vomiting, cough with mucous sputum, and has been sick in bed.

Two days ago she began to have pain in the right side of the chest and dyspnœa.

Now, on the third day of the pneumonia, the tongue is coated and dry; the urine 1020, contains a large quantity of albumin and red blood cells. Over the right lower lobe there is dulness on percussion and broncho-vesicular breathing; there are coarse râles over both lungs.

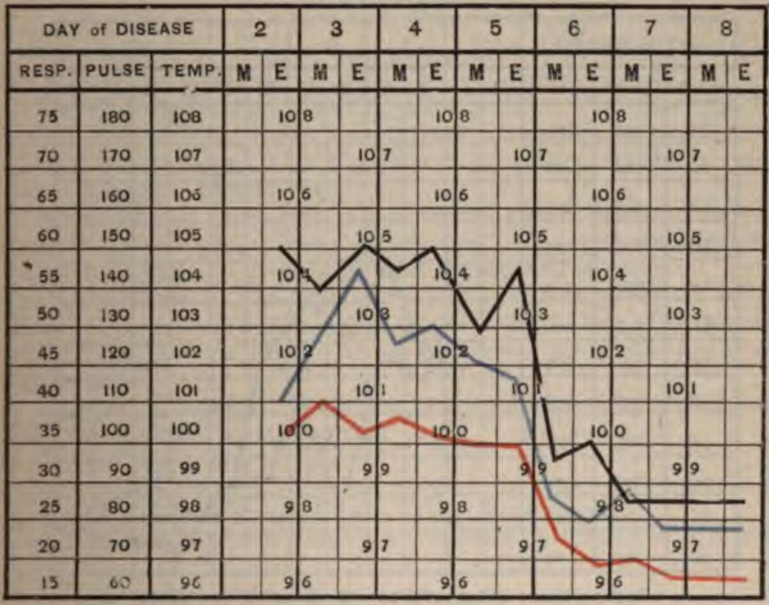
On the ninth day the urine contained only a trace of albumin. The temperature fell gradually from the seventh to the eleventh day and she recovered.

(For Chart of Case XXXVI, see Fig. 55.)

CASE XXXVII. *Lobar Pneumonia; General Bronchitis.*—Male, 20 years old, November 14, 1894.

Three days ago he began to be feverish and had pain in the small of his back.

FIG. 56



Temperature black, Pulse red, Respiration blue.

CASE XXXVII.—LOBAR PNEUMONIA; INFLUENZA; BRONCHITIS.

To-day he began to cough, with mucous expectoration, and had pain in the right side of his chest.

Now, on the third day, over both chests there is sibilant breathing and coarse râles. Over the right chest in front, over the first two interspaces, there is dulness on percussion, bronchial voice and breathing, and crepitant râles.

On the fourth day the expectoration was profuse and bloody.

On the fifth day the bloody expectoration continued and he was delirious.

On the sixth day there was still a large quantity of serous blood-stained expectoration.

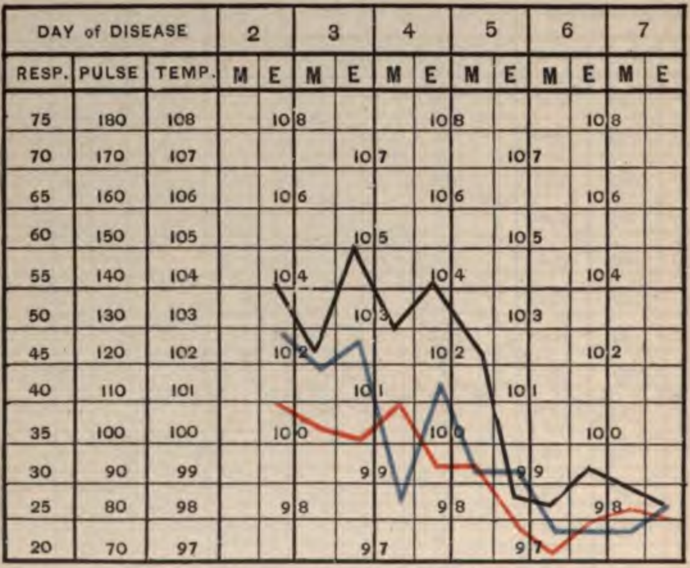
On the seventh day he was much better and was eating solid food.

On the eighth day the pneumonia had resolved and there was very little bronchitis left. He has been treated with ipecac, aconitine, and digitaline.

On the fourteenth day he was discharged cured.

(For Chart of Case XXXVII, see Fig. 56.)

FIG. 57



Temperature black, Pulse red, Respiration blue.

CASE XXXVIII.—LOBAR PNEUMONIA; INFLUENZA; BRONCHITIS.

CASE XXXVIII. *Lobar Pneumonia; Acute Bronchitis; Influenza.*—Female, 21 years old, November 14, 1894.

Two weeks ago she had headache, vomiting, fever, and cough with mucous expectoration. These symptoms continued until last night, when she became much worse, with a severe chill, dyspnœa, and the expectoration became bloody.

Now, on the fifteenth day of the influenza and the second of the pneumonia, she is coughing up large quantities of muco-purulent, bloody sputum. Over the left lung behind, from the middle of the scapula to the base, there is dulness on percussion, bronchial voice and breathing, and subcrepitant râles.

On the third day she continued to cough up large quantities of muco-purulent sputum mixed with a good deal of blood.

On the fifth day she defervesced.

After this the expectoration steadily diminished and she improved.

She was treated with aconitine and digitaline.

On the thirtieth day of her influenza, and fifteen days from the beginning of her pneumonia, she was discharged cured.

(For Chart of Case XXXVIII, see Fig. 57.)

CASE XXXIX. *Lobar Pneumonia; Bronchitis; Death.*—Male, 28 years old, December 22, 1894.

The man had been habitually intemperate. Two days ago he had a chill lasting for ten minutes. After this he had fever, cough with mucous and bloody expectoration, dyspnœa, and pain over both sides of the chest.

Now, on the third day, all over the chest there is sibilant breathing and coarse râles. Over the left lower lobe behind there is dulness on percussion and diminished breathing. Over the third, fourth, and fifth interspaces in front there is bronchial voice and breathing and subcrepitant râles. The bloody expectoration continues; he is delirious and continued so throughout.

He was treated with aconitine and digitaline, tincture of nux vomica, and cardiac stimulants.

On the eighth day he died.

Autopsy.—There is recent fibrin over the whole left lung. The entire left lung is in a condition of red hepatization.

The bronchi of both lungs are congested and filled with muco-pus.

The heart is normal.

The kidneys are in a condition of acute degeneration.

(For Chart of Case XXXIX, see Fig. 58.)

CASE XL. *Lobar Pneumonia beginning on the Thirteenth Day of a Severe Influenza.*—Male, 55 years old, January 6, 1890.

To-day he began to have coryza, conjunctivitis, cough with a little sputum, and felt sick.

Four days later he felt worse and went to bed. He had general pains and was mildly delirious. He remained in bed, dull and drowsy, but not looking very badly, with no physical signs over the lungs, until the thirteenth day of the influenza, when his pneumonia began. He had pain in the left chest and cough with profuse muco-purulent sputum; urine 1026, no albumin.

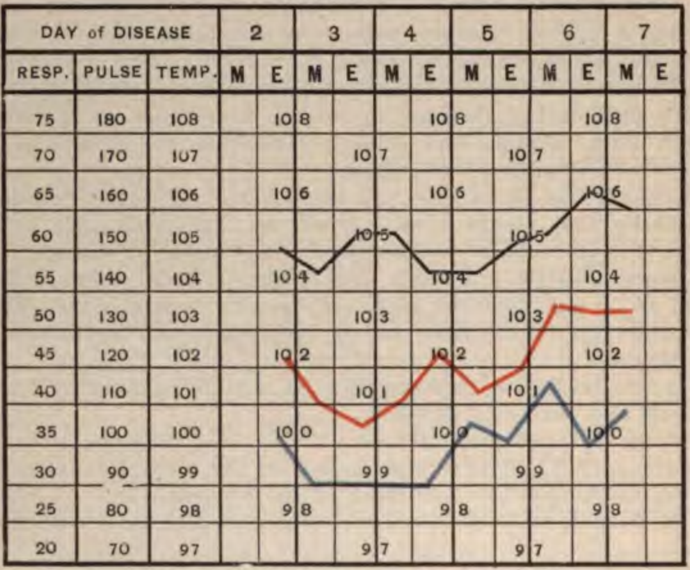
On the second day of the pneumonia, over the left lung in front there is dulness on percussion, bronchial breathing, and a few râles.

He grew steadily worse, with feeble heart, venous congestion, and embarrassed breathing.

On the fifth day of the pneumonia he died.

Autopsy.—In the heart there is a little old thickening of the aortic and mitral valves.

FIG. 58



Temperature black, Pulse red, Respiration blue.

Case XXXIX.—Lobar Pneumonia; Influenza; Death.

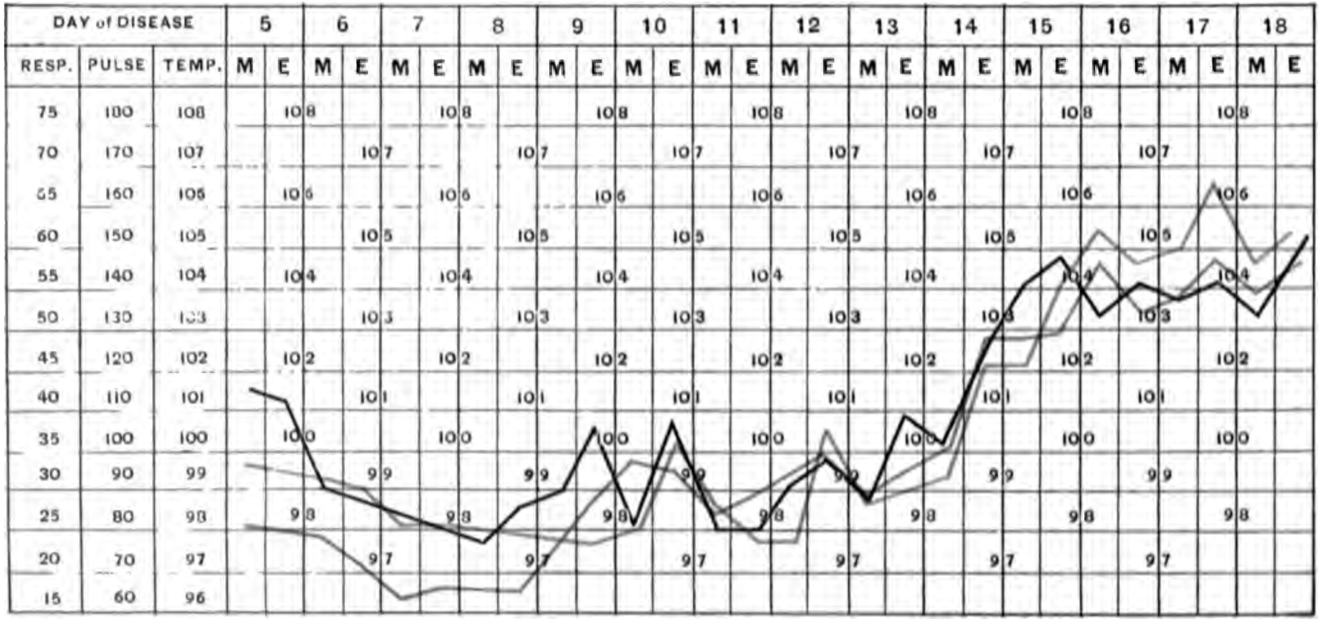
The left lung is covered with old pleuritic adhesions. There is recent fibrin over the upper lobe. The bronchi are congested and full of pus. There is nearly complete hepatization of the upper and lower lobes. In the right lung the bronchi are full of pus; the lower lobe is congested.

The spleen, liver, and kidneys are normal.

(For Chart of Case XL, see Fig. 59.)

Case XLI. *Pneumonia; Influenza; Obscure Physical Signs.*—Male, 23 years old, March 2, 1900.

FIG. 50



Temperature black, Pulse red, Respiration blue.

CASE XL.—LOBAR PNEUMONIA; INFLUENZA; DEATH.

Nine days ago he began to have pains all over the body, with a cough. He remained in bed one day and then went to work, feeling sick and coughing.

Three days ago, on the seventh day of the influenza, he had chills, headache, vomiting, severe pain in the right side, high fever, and felt very sick. For the past two nights he has been delirious.

Now, on the fourth day of his pneumonia, his face is flushed and anxious, tongue coated, pulse 100 and full, temperature 103°-104° F., breathing shallow; urine 1021, a trace of albumin. There are no physical signs, except a little dulness in the right axillary region.

On the fifth day of the pneumonia the mind was clear; all the symptoms were abating; the sputum was rusty. Hæmoglobin 100 per cent, red cells 5,000,000, white cells 10,000. He defervesced in the afternoon.

On the eleventh day he was discharged cured.

Case XLII. *Lobar Pneumonia with Influenza, Long Duration.*—The lower lobe of the left lung was hepatized and resolution was completed on the sixteenth day. The right lower lobe was hepatized on the sixteenth day and resolved on the twenty-fourth day. The left pleural cavity was one-fourth full of serum on the thirty-first day. The temperature did not fall to the normal until the fifty-second day.

The patient finally recovered.

(For Chart of Case XLII, see Fig. 60.)

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Duration of the Disease.—I have shown in Figure 32 the ordinary duration of the disease in the cases that terminate by crisis. In Figure 61 is shown the ordinary duration of the fatal cases. The chart shows in the broken line the duration of 74 fatal cases reported by Dr. Stevens, of Edinburgh; in the straight line the duration of 149 fatal cases observed by myself.

In the fatal cases of lobar pneumonia there seem to be three main factors in causing death—the pneumococcus sepsis, the inflammation of the lung, and the existence of complications. These three factors act separately or together.

The pneumococcus sepsis may cause death with comparatively little inflammation of the lung, the patients dying with all the symptoms of general poisoning.

The inflammation may involve the whole of one lobe, and yet

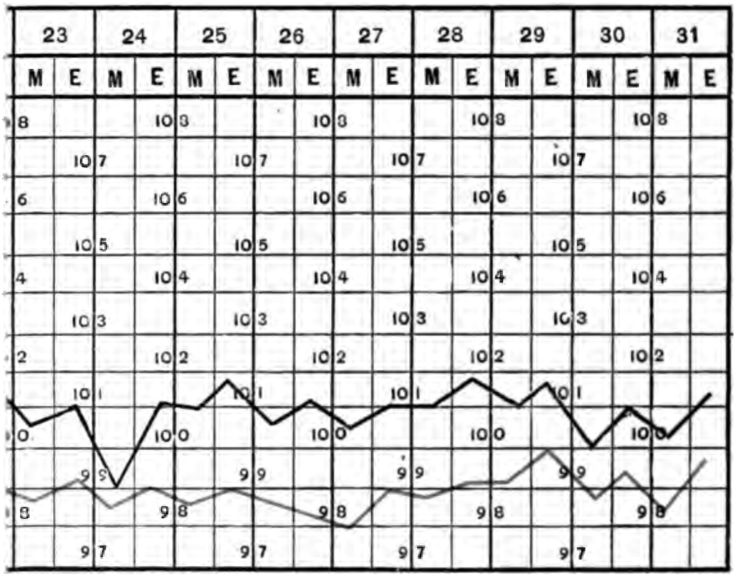
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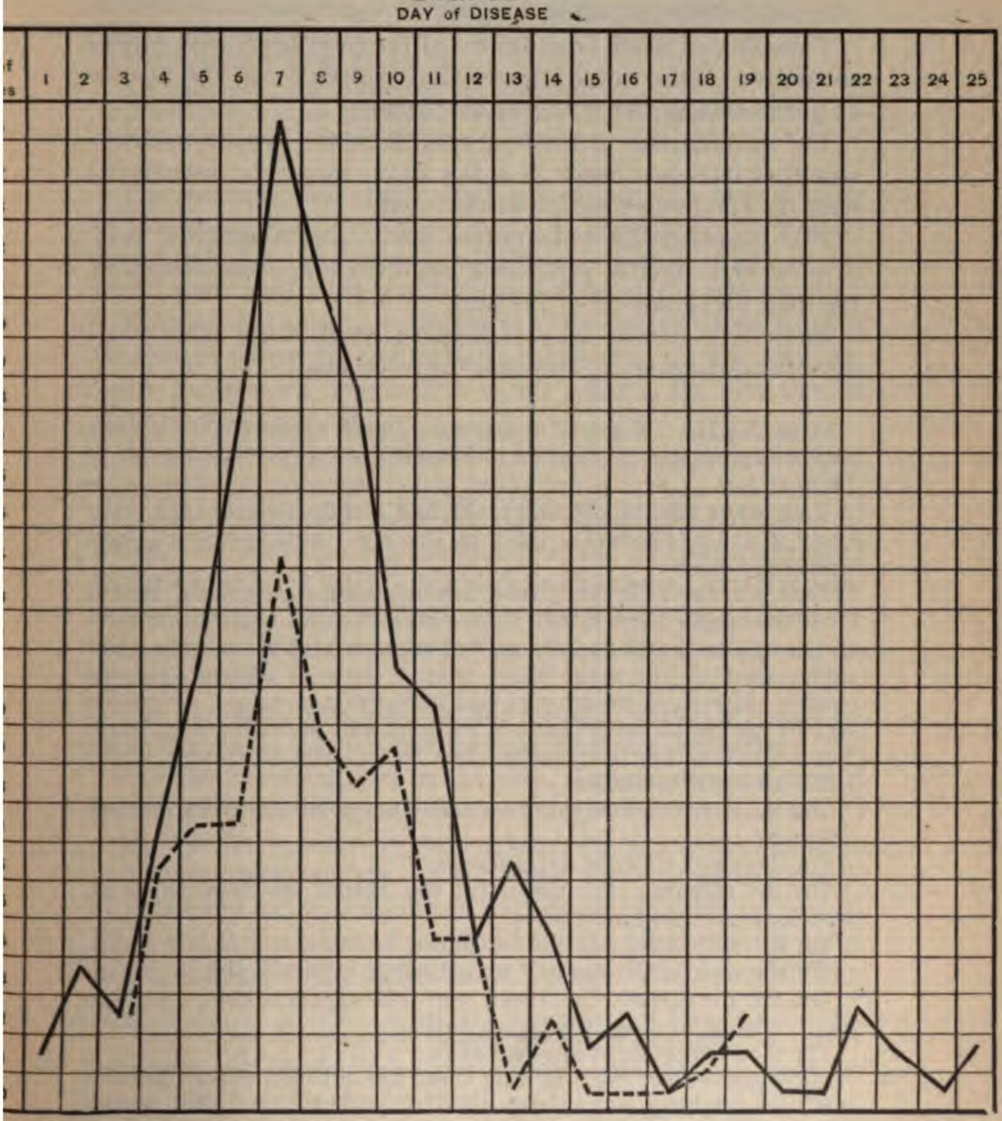
FIG. 61



Duration of Fatal Cases.

it would seem as if the patients could get well were it not for the effect of the pneumococcus toxin on the right heart.

This effect of death from failure of the right heart with venous congestion of the lungs is especially common when the poisons of pneumonia and of influenza act together.

The inflammation of the lung may, from the first, be so extensive that it causes death in a few days, simply by interfering with the breathing function of the lungs.

The complications which cause death are: meningitis, pericarditis, endocarditis, peritonitis, nephritis, empyema, abscess of the lung, and gangrene of the lung.

There is a curious class of cases in which death takes place after defervescence and commencing resolution.

CASE XLIII. *Lobar Pneumonia; Death after Defervescence and Commencing Resolution.*—Female, 50 years old, March 3, 1888.

Four days ago, in the night, she had a chill, fever, cough with rusty sputum, headache, pain in the right side, dyspnœa, and vomiting.

Now, on the fifth day, face flushed, tongue dry and coated. Over the upper part of the right chest in front there is dulness on percussion, harsh breathing, and subcrepitant râles. Over the upper part of the right chest behind there is dulness on percussion, bronchial breathing, and subcrepitant râles. Over the rest of the right lung there is sibilant and sonorous breathing. The heart's action is irregular, but there is no murmur. Urine 1022, albumin, no casts.

On the seventh day she was delirious, cyanotic, with marked dyspnœa.

On the ninth day she defervesced.

On the eleventh day the pulse was feeble, she was delirious, vomiting, and uncomfortable.

On the thirteenth day she continued to vomit and looked badly.

On the eighteenth day the temperature began to rise.

On the nineteenth day there were subcrepitant râles over the right lung behind, as if with resolution.

On the twenty-third day she died.

Autopsy.—Over the left lung there are old pleuritic adhesions.

The right lung is coated with fibrin. The lower part of the upper lobe and the upper part of the lower lobe are congested, partly aërated, and look like lobar pneumonia in the stage of resolution.

The left ventricle of the heart is hypertrophied and there is a little old thickening of the aortic and mitral valves.

The liver is large.

The spleen is large and soft.

The stomach and intestines are normal.

The kidneys show the lesions of acute degeneration.

(For Chart of Case XLIII, see Fig. 62.)

The mortality from pneumonia is a considerable one, ranging in hospitals from 12 to 34 per cent. It seems to be the general impression that the disease is more fatal now than it was a number of years ago, but it is difficult to determine this accurately.

Treatment.—Lobar pneumonia is a disease for which there is no routine treatment applicable to every case; on the contrary, much judgment is required to decide what is the best way of managing each patient.

As a rule, all the patients are to be kept in bed and on a fluid diet until the temperature has fallen to the normal and the exudate has disappeared from the lung. But there are cases in which, even while the inflammation is active, a little scraped beef improves the heart action; and in patients in whom defervescence or resolution is delayed for an unusually long time it may be wise to give solid food and to allow the patient to sit up before the usual time.

The headache, sleeplessness, and general discomfort may require the use of opium, sulphonal, the bromides, or chloral.

For the excessive pain in the side, large poultices, repeated every two hours, and hypodermics of morphine are to be used.

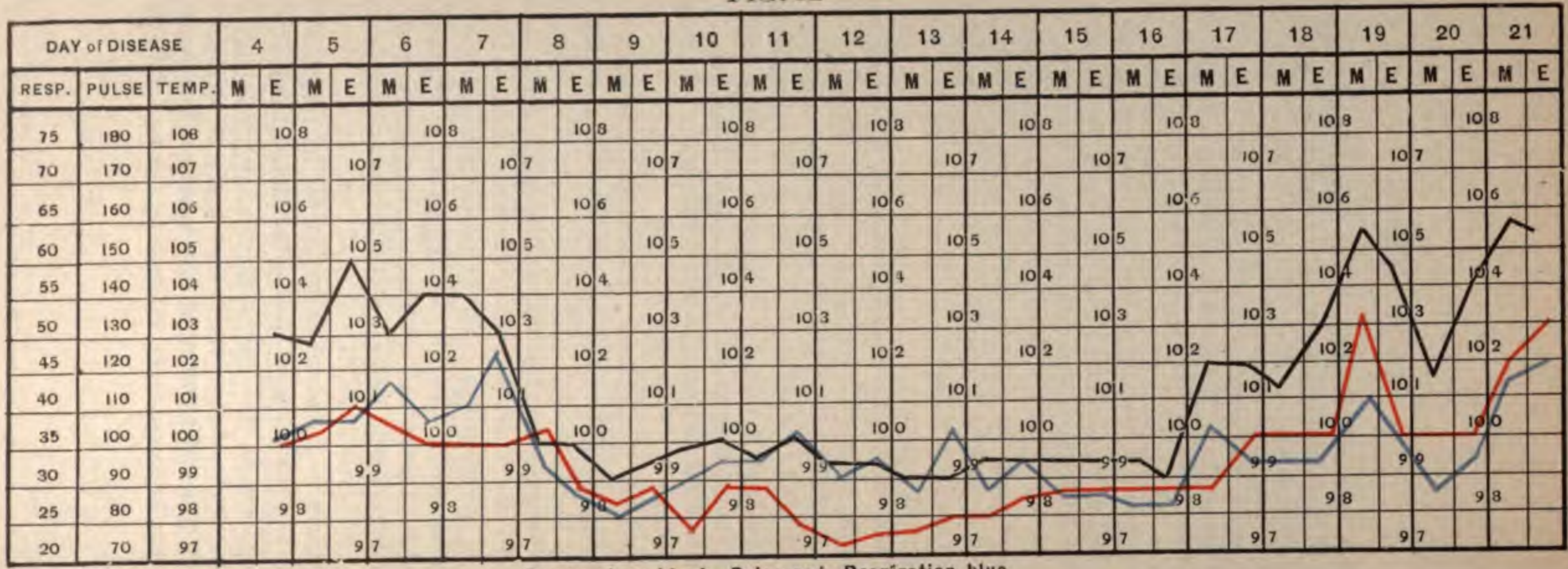
For the feeble heart we employ alcoholic stimulants, digitalis, caffeine, or strophanthus. A very good combination is 5 grains of potassium iodide, 1 minim of fluid extract of digitalis, and 20 minims of fluid extract of convallaria, given together every three hours, or hypodermic injections of 5 minims of tincture of digitalis.

When there is excessive congestion of the lungs, with great dyspnœa, and many coarse and subcrepitant râles all over the lungs, relief can be obtained by repeated dry cupping, by hypodermic injections of one-fiftieth of a grain of nitroglycerin, or by 20-drop doses of tincture of nux vomica. The cyanosis and dyspnœa can be temporarily relieved by the use of oxygen.

1. Liq. Peptonoids 1/2 ℥ q 3 h. Regardless of other diet. —

[illegible] 36 drops a minute [illegible] 17

FIG. 62



Temperature black, Pulse red, Respiration blue.

CASE XLIII.—LOBAR PNEUMONIA; DEATH AFTER DEFERVESCENCE.

If there is a good deal of general catarrhal bronchitis, the patients can be benefited by dry-cupping or by the use of one-tenth of a grain of ipecac every hour.

For the exudative inflammation of the lungs the plans of treatment most frequently adopted are:

Venesection employed once or repeated several times, the quantity of blood taken to be considerable. This plan is not often employed at the present time.

Large doses of calomel, 12 to 30 grains, placed dry on the tongue—from one to four such doses. This plan seems to answer well for some cases, to be of no use in others, and is attended with risk of producing salivation.

Small doses of calomel, ¼ to 1 grain, given every hour up to six doses. This seems to be of moderate efficacy.

Drachm doses of magnesium sulphate, given every hour up to eight doses. This, again, does not seem to be of much value.

The employment of either tincture of aconite or tincture of veratrum viride, in doses of from 2 to 5 drops, at first every hour and later at longer intervals, has given good results in the hands of some physicians.

Cold affusions to the chest, cold baths, blisters, the antipyretic drugs, quinine, and carbonate of ammonium have all been much used, but are all of doubtful efficacy.

The treatment of the exudative inflammation which seems to me the most satisfactory is that by the combined use of aconitine, digitaline, and whiskey. The aconitine and digitaline used are those made by Merck. The dose is 1-6 mgm. (gr. 1-400) of aconitine and 1-8 mgm. (gr. 1-500) of digitaline in tablets; the two drugs are given together. The frequency of the dosing is regulated by the pulse. We endeavor to get a pulse of about 80 and of good quality. In order to do this it may be necessary to give the drugs every fifteen minutes, every half hour, every hour, or every two hours, as the case may be. If the pulse be slow enough, but too feeble, whiskey must be given at regular intervals. It is not safe to follow this plan of treatment unless the patient can be watched. If it be carried too far, the pulse may fall to 30 and the patient pass into a condition approaching collapse. This plan of treatment does not seem to be of any

✓ Abbott

service in the cases in which the symptoms of poisoning are out of proportion to the extent of lung inflamed, nor in the cases which are complicated by pericarditis.

repeated

Carbonate of creosote has been used a good deal during the last few years and is liked by some physicians.

The whole behavior of the disease is such as to give one the feeling that it ought to be successfully treated by an antitoxin. But so far a satisfactory serum has not been manufactured, and reports of a few successful cases are of no value.

For example, in 1896 I used one of the antistreptococcus serums in a number of cases of pneumonia. In the first four patients the results were rather striking. The patients all had temperatures up to 105° F.; in three of them the pulse reached 120, the respiration was from 40 to 60; two of the patients were delirious, all looked very seriously ill. Each of them received hypodermic injections of 2 ounces of the serum twice or three times in each twenty-four hours. Two of the patients defervesced on the sixth day, and two on the seventh day of the pneumonia. The results seemed to be satisfactory, but later in the winter I gave up the use of the serum; it evidently did nothing.

The rules which I follow in the treatment of lobar pneumonia are:

1. There are cases, some of them mild, some of them severe, in which there is no straightforward indication for treatment. In these patients I look after the nursing and give no medicine.

2. In many cases headache, pain in the side, sleeplessness, and the feeling of illness call for relief. For this purpose opium is altogether the most efficient drug. By combining morphine with acetanilid the sedative effects of the morphine are very much increased. For persons who cannot take opium we have to do the best we can with the bromides and the antipyretics of the coal-tar group.

3. There are patients in whom from the first there is an extensive catarrhal bronchitis, attended with a large production of mucus, more or less bloody, from the bronchi, and venous congestion of the bronchi. While the ordinary remedies for bronchitis are of service, it is important also to employ efficient and repeated dry-cupping, to try to accelerate the circulation of the

217

blood in the walls of the bronchi by digitalis, convallaria, and hypodermics of nitroglycerin, and to diminish the excessive production of mucus by the tincture of nux vomica. (?)

4. There are cases to which seems applicable the abortive treatment by large doses of calomel. I see this done by other physicians, sometimes with good results. It is a long while since I have used it myself.

5. In a great many cases there is failure of the heart's action, which calls for treatment. This failure, however, is not always exactly of the same character.

(*a*) There is a heart failure of the same character as that which we get in any of the severe infectious diseases. It requires the ordinary management with alcoholic and cardiac stimulants. I like in these cases the combination of iodide of potash, 5 grains, fluid extract of digitalis, 2 minims, and fluid extract of convallaria, 10 minims, every three hours, or hypodermic injections of tincture of digitalis. p 215

(*b*) In some patients quite early in the disease the pulse is at 120 and over, and the breathing at 30 and over, without an excessive production of mucus in the bronchi or of serum in the lungs. These patients can be much improved by small doses of aconitine and digitaline with more or less whiskey.

The particular combination which has seemed to me to be the most reliable is that of aconitine, 1-6 mgm.; digitaline, 1-8 mgm.; and whiskey in doses from one drachm to one ounce. The digitaline and aconitine are given together at intervals of from one to three hours, the whiskey every three or four hours. The guide for the frequency of the use of these drugs is the effect on the circulation. We try to keep a pulse of between 90 and 100 and of good quality; with this the breathing will be better. If, however, we do not get the effect of slowing the pulse within about twenty-four hours, there is no use in continuing this plan of treatment.

6. At about the fourth day of the pneumonia we find only too often that the bronchi and lungs are beginning to fill up with mucus and serum, with coarse and subcrepitant râles all over both lungs. This is a most unfavorable symptom. When it begins, and even after it has continued for some time, the radial pulse is

often good. The failure seems to be on the part of the right heart. For this condition combinations of digitalis, convallaria, and nux vomica (as much as 20 minims of the tincture of nux vomica at a dose); hypodermic injections of nitroglycerin; dry cups over the chest; and the inhalation of oxygen, seem to do the most good.

7. An excessive distension of the intestines with gas is an evidence of virulent poisoning, and it may interfere seriously with the action of the heart and lungs. For this condition irrigation of the rectum with large quantities of hot water may be of much service.

8. In cases which show a disposition to become protracted it is often wise to give scraped beef before the temperature has fallen to the normal, and to get the patients out of bed before the lung has resolved. But both of these must be done with judgment.

SECONDARY LOBAR PNEUMONIA.

This is an exudative inflammation involving one or more lobes of one or both lungs, occurring in persons already suffering from some disease or injury, and due to infection with streptococci or pneumococci.

Persons who are confined to bed by infectious diseases, by injuries or inflammations of the brain or spinal cord, by surgical operations, or by severe injuries are liable to have venous congestion of the dependent portions of the lungs and to inhale streptococci or pneumococci. In this way they often contract either a true broncho-pneumonia or a pneumonia which somewhat resembles a lobar pneumonia.

Morbid Anatomy.—The inflammatory process involves irregular areas of one or of both lungs. We find these areas after death in the condition of red or gray hepatization and surrounded by congested lung, but no complete consolidation of an entire lobe. The inflammation is of exudative type, with fibrin, pus, epithelium, and streptococci or pneumococci in the air spaces and small bronchi.

Symptoms.—In many cases the pneumonia can hardly be said to give its rational symptoms or physical signs. We find the lesion after death, but are not certain of its existence during life. In some cases, however, there are chills, fever, rapid breathing, pain, cough, and expectoration, with the physical signs of bronchitis or of consolidation of small portions of the lung.

The treatment of such a pneumonia is unsatisfactory.

Lobar Pneumonia with the Formation of Connective Tissue.

It is well known that in some forms of inflammation of the lung there is a production of new connective tissue around the bronchi and blood vessels, in the septa between the lobules, and in the walls of the air spaces. It is not so well known that in these same forms of pneumonia there may be also a production of new connective tissue in the cavities of the air spaces and of the small bronchi. This new tissue either grows directly from the walls of the air spaces or is formed out of plugs of coagulated matter and of cells which are formed within their cavities.

Such a productive pneumonia has been recognized under a variety of names—gray induration, fibroid induration, cirrhosis, interstitial pneumonia, chronic pneumonia, desquamative pneumonia, parenchymatous pneumonia, etc. If we look over all the different lungs in which such a productive pneumonia has been developed, we find that they can be classified as follows:

1. A productive pneumonia associated with the growth of tubercle bacilli.
2. A productive pneumonia associated with the growth of actinomyces.
3. A productive pneumonia due to the inhalation of particles of coal or of stone.
4. A productive pneumonia caused by constitutional syphilis.
5. A productive pneumonia secondary to changes in the pleura.
6. Broncho-pneumonia.
7. A special form of lobar pneumonia.

It is concerning this last variety of productive pneumonia that our information is the least exact, and it is to this variety that I wish especially to call attention. The ordinary belief has been that it is possible for a regular exudative lobar pneumonia, instead of resolving, to be succeeded by a chronic productive inflammation. I believe, on the contrary, that a regular exudative lobar pneumonia terminates only in resolution or in death, and that lobar pneumonia with the production of new connective tissue is from the first a special form of inflammation of the lung. My reason for this belief is that I have seen a number of lungs which seem to show the different stages of the inflammatory process.

The literature on the subject is not very abundant. Charcot[1] describes this condition as following one or more attacks of ordinary lobar pneumonia. Coupland[2] gives a very good description with drawings, and believes that the new tissue is formed from the intra-alveolar exudation of ordinary lobar pneumonia. Kidd[3] describes two cases with a subacute history which he regards as cases of lobar pneumonia terminating in induration. Buhl[4] considers the disease to be a primary one, which runs a subacute course and has nothing to do with ordinary pneumonia. Heitler[5] gives an account of the disease as observed in five cases. The development of the disease, he says, is more or less acute, with fever, dyspnœa, cough, prostration, sometimes rigors; the constitutional depression is much less marked than in acute pneumonia. The fever is irregular and not over 102° F. The sputum is mucous, muco-purulent, or fetid. In two of the cases there was retraction of the wall of the thorax. The consolidation involved in three cases the right upper lobe, in one case the whole right lung, in one case the lower right lobe. The hepatization was smooth, with necrotic and cheesy areas and cavities. The course of the disease was subacute, but with an acute invasion, lasting from fifty days to nine months and nineteen days. Wagner[6] has described six cases apparently belonging to this group, running a subacute course with retraction of the wall of the chest, but terminating in recovery.

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[1] Rev. Mens. de Med. et Chir., 1878, p. 776.

[2] Trans. Lond. Path. Soc., vol. xxx., p. 224.

[3] Lancet, April 5, 1890.

[4] Buhl, Briefe, p. 47.

[5] Wiener med. Wochenschrift, 1884 and 1886; v. Kahlden, Ziegler's Beiträge, xiii., p. 279.

[6] Deutsch. Arch. f. klin. Med., vol. xxxiii.

I have seen thirteen cases which seem to belong to this group, and to demonstrate that there is a form of lobar pneumonia which is, from the outset, anatomically distinct from the ordinary form. It is from the first an exudative inflammation with the production of new tissue, not a simple exudative inflammation. Such an inflammation naturally lasts longer and is more likely to become chronic than is the case with a simple exudative inflammation. I can see no reason to believe that in ordinary lobar pneumonia the pus and fibrin are ever replaced by connective tissue.

The development of the lesion seems to be as follows:

1. Congestion of the lung; exudation of serum, fibrin, and pus into some of the air spaces; the formation in other air spaces of irregular plugs with prolongations from one space into others, the plugs composed of a nearly homogeneous or finely-fibrillated material, none of them large enough to fill or distend the air spaces; a swelling and thickening of the walls of the air spaces, with a very considerable increase in the number of epithelial cells which cover them; more or less general catarrhal bronchitis; fibrin on the pulmonary pleura.

2. New cells, of the type of connective-tissue cells, are formed in the plugs; the walls of the air spaces are more swollen, and may be infiltrated with small round cells; new blood vessels are formed in the plugs, which can be artificially injected from the pulmonary vessels. The gross appearance of the lung at this time is usually characteristic. One or more lobes are consolidated; they are not large as in ordinary pneumonia; their color is red or gray; the cut surface is smooth, not granular.

3. The growth of new connective tissue within the air spaces, in their walls and along the arteries and bronchi is so extensive that many of the air spaces are obliterated. The surface of the lung is now covered with connective-tissue adhesions; the bronchi contain muco-pus; the lung is red, mottled with white or gray or black; it is dense and hard; portions of it may be necrotic or cheesy or broken down into cavities.

Five of my cases illustrate the first period of the development of the lesions:

Case I.—The duration of the disease was ten days. The entire left lung was consolidated, small, smooth, of gray color, with fibrin coating the pulmonary pleura. The right lower lobe was partly hepatized and red. The walls of the air spaces were thickened and coated with epithelial cells. There was a growth of new connective tissue around the blood vessels and bronchi. The air spaces contained small anastomosing plugs of a nearly homogeneous matter.

Case II.—The duration of the disease was seventeen days. The left lower lobe was consolidated, small, red, and smooth, its surface covered with old adhesions. The walls of the air spaces were thickened; their cavities contained the plugs already described.

Case III.—The duration of the disease was fourteen days. There was a general bronchitis. The right upper lobe was consolidated, of reddish-gray color, its pleura coated with fibrin. The walls of the small bronchi were thickened and infiltrated with cells. The walls of the air spaces were thickened and coated with epithelium. Some of the air spaces contained pus and fibrin, others the plugs already mentioned.

Case IV.—The duration of the disease was seven days. The left lower lobe presented the regular picture of the red hepatization of ordinary pneumonia. The left upper lobe was consolidated, small, smooth, and red. The walls of the air spaces were thickened and coated with epithelium; their cavities contained pus and fibrin or the plugs.

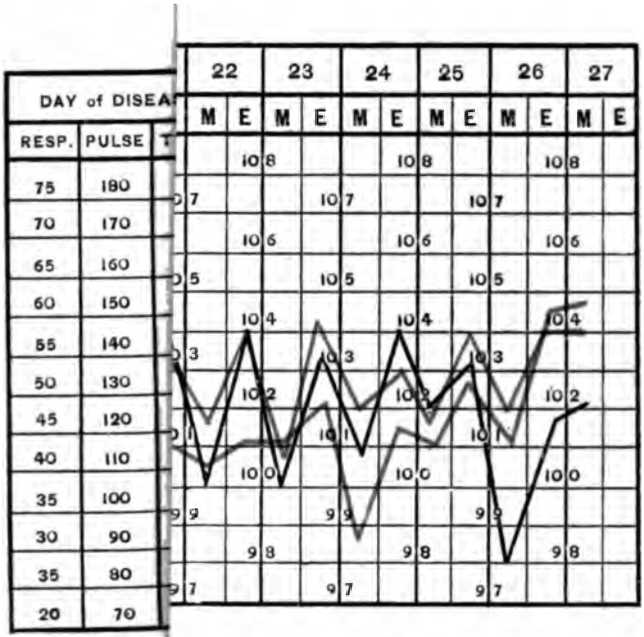
Case V. *Lobar Pneumonia with the Formation of New Connective Tissue.*—Male, 36 years old, January 31, 1895.

Three days ago the man had a shaking chill, vomiting, pain in the right chest, cough and rusty sputum.

Now, on the fourth day, over the upper lobe dulness on percussion, bronchial voice and breathing, and crepitant râles. Urine 1017, a little albumin.

The man continued to have fever, to cough, and gradually to lose strength until he died on the twenty-eighth day.

Autopsy.—The left pleural cavity is full of clear serum. There are many old adhesions over the upper lobe. The pulmonary pleura is covered with fresh fibrin. The bronchi contain muco-pus. There is some irregular, smooth, white hepatization.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The entire right lung is in the condition of smooth, white hepatization.

The left ventricle of the heart is dilated and hypertrophied, the muscle is fatty.

The spleen is large and soft.

The kidneys are large, with thick cortex.

(For Chart of Case V, see Fig. 63.)

Six of my cases illustrate the second period of the development of the lesion.

CASE VI.—Duration, nineteen days. The right upper lobe was consolidated, smooth, and red. There was a growth of new connective tissue around the arteries, in the septa, and in the walls of the air spaces. Some of the air spaces contained epithelium, others plugs of the same shape and appearance as seen in the preceding group of cases; but there were, in addition, connective-tissue cells embedded in the basement substance composing the plugs.

CASE VII.—Duration, sixteen days. The right middle and lower and left lower lobes were consolidated, small, and smooth. The walls of the air spaces were thickened. Some of their cavities contained fibrin and pus, others plugs of connective tissue containing blood vessels.

CASE VIII.—Duration, twenty-three days. The left lower lobe was consolidated, small, and gray. The walls of the air spaces were thickened; they were covered with epithelium and contained plugs of connective tissue.

CASE IX.—Duration, six days. General bronchitis. The left upper lobe was consolidated, large, and red. The walls of the air spaces were thickened. Some contained pus and fibrin, others plugs of connective tissue.

CASE X.—Duration, thirteen days. The left lower lobe was in the condition of ordinary red hepatization. The right upper lobe looked like the resolution of an ordinary pneumonia, and some of the air spaces contained degenerated exudation, but in others there were plugs of connective tissue.

Two cases illustrate the third period of development of the lesion:

CASE XI.—Duration, fifty-two days. The left lung was covered with old adhesions, consolidated, hard, smooth, mottled red and white, small. The growth of new connective tissue in the walls of the air spaces and in their cavities had nearly obliterated the natural structure of the lung.

CASE XII.—Duration, fifty-one days. The pleura was thickened and coated with fibrin. The left pleural cavity was half full of serum. The left upper lobe was consolidated and of a pinkish-white color. It was almost entirely changed into connective tissue.

Etiology.—In three of the cases there was a distinct history of prolonged exposure to cold and wet. In one case the symptoms followed immediately after the patient's having fallen into an excavation. In one case, for twenty days before the initial chill the patient was miserable and had a troublesome cough. In one case, for twenty-one days before the initial chill the patient suffered from headache, loss of appetite, and prostration. In one case, under observation throughout, there was an attack of lobar pneumonia terminating in resolution after eleven days; the patient was discharged from the hospital well, and after an interval of eighteen days came the beginning of the fatal attack in the other lobe of the same lung. In one case the patient stated that he had an attack of pneumonia five years before, and that for one year he had been troubled with cough and muco-purulent expectoration.

Symptoms.—In ten of the cases the invasion of the disease was marked by chills and a rapid rise of temperature. There was cough in all the cases, the sputa rusty in five cases, muco-purulent in two cases, bloody in one case. The temperature was rarely over 104° F., and in some of the cases not over 100° F. In some of the cases delirium is noted as a prominent symptom. One case was supposed to be acute phthisis, one acute meningitis, and one acute general tuberculosis. Three of the patients passed fairly into the typhoid condition. The physical signs of the consolidation of the lung were well marked, except in one case. The duration of the disease was for six, seven, ten, thirteen, fourteen, sixteen, seventeen, nineteen, twenty-three, twenty-eight, fifty-one, and fifty-two days—in most of the cases longer than that of an ordinary lobar pneumonia.

There seems to be, therefore, a form of lobar pneumonia which is anatomically different from the regular form. Its physical signs are, of course, the same, but its clinical symptoms are somewhat different. Although the patients have the same chill, fever, cough, expectoration, and pain as in the regular cases, yet there is something about the course of the disease which makes its diagnosis possible even during its early days. The temperature does not run high, but the tendency to cerebral symptoms and the typhoid state is very marked, and most of the cases run a protracted and subacute course. Concerning the prognosis it is not possible to speak certainly, but there seems no reason why recovery should not be possible.

BRONCHO-PNEUMONIA.

Definition.—An infectious inflammation, with exudation from the blood vessels, a formation of new connective tissue and the growth of pathogenic bacteria, which involves principally the walls of the bronchi and those of the air spaces which surround the inflamed bronchi.

Synonyms.—Capillary bronchitis; Lobular pneumonia; Catarrhal pneumonia.

There seems to be no form of pneumonia which does not have associated with it more or less bronchitis, so that every pneumonia is in one sense a broncho-pneumonia. But it has long been recognized that there is one form of inflammation of the lung which is different from others, and in which the share of the bronchi is especially important. For several reasons, however, the popular notions concerning the disease have become somewhat confused. It was seen that in some cases, while there was a bronchitis extending down to the small bronchi, there was no consolidation of the lung, and so these were called cases of capillary bronchitis. It was seen that there were cases of general bronchitis with consolidation of circumscribed portions of the lung, and it was inferred that the inflammation extended from the bronchi to the air spaces which empty into them; so these were called cases of lobular pneumonia. This, however, was an error in observation. Areas of atelectasis do correspond to bronchi, but the areas of consolidation do not so correspond. It was seen that in some cases the symptoms and lesions could not be distinguished from those of phthisis, and it was not understood that the only real difference between the two was the presence or absence of the tubercle bacilli. Numbers of curious ideas were connected with the term "catarrhal inflammation," and it was not realized that a catarrhal inflammation is nothing but an exudative inflammation occurring in a mucous membrane.

It is to be regretted that the erroneous idea still exists that a broncho-pneumonia is simply an inflammation of the bronchi which extends to the air spaces opening into these bronchi.

Etiology.—Broncho-pneumonia is the ordinary pneumonia of children; it is frequently seen in young persons, and occasionally in adults and old persons.

It occurs as a primary inflammation, is often secondary to measles, whooping cough, and diphtheria, less frequently to the other infectious diseases. Persons confined to bed by injury or disease and persons with emphysema are liable to subacute forms of broncho-pneumonia.

The disease is most frequent during the cold and wet months of the year; in some cases there is a history of exposure to cold; in others no exciting cause is discoverable. Children crowded together in asylums are especially liable to the disease.

The same patient not infrequently suffers from several attacks of broncho-pneumonia.

The ordinary bacteria present are pneumococci or streptococci. It is important to remember that the distinction between lobar and broncho pneumonia is not in the infection, for that is produced by the same pathogenic organisms in both. The difference is in the anatomical characters of the inflammation which is produced by these organisms. The anatomical characters of the inflammation seem to be determined by the age of the patient. In children the walls of the bronchi are most easily irritated; in adults, the air spaces. So we find with the same infections that in children we regularly have broncho-pneumonia, in adults lobar pneumonia, and in adolescents either one of these inflammations.

Morbid Anatomy.—In persons who die from broncho-pneumonia the lungs after death present considerable variety in their gross appearance. The mucous membrane of the trachea and large bronchi may appear to be normal, or is congested and coated with mucus, or the small bronchi may contain pus. The walls of the small bronchi are thickened so that a section of the lung looks studded with little nodules. In some cases most of the small bronchi of both lungs have their walls thickened in this way; more frequently it is only the bronchi of one lung or of one lobe. Around the bronchi whose walls are thickened are zones of consolidated lung from the size of a pinhead to that of a pea. Scattered through one or both lungs are irregular areas of consolidation having no definite relation with the bronchi. They may be so large and numerous that an entire lobe or an entire lung is completely consolidated. The pulmonary pleura may be coated with fibrin. In the lungs of very young children there may be consolidated shrunken portions due to collapse of the air vesicles—the so-called areas of atelectasis. The same condition is found with bronchitis, and in children who die so soon after birth that the whole of the lungs has not become aërated. The bronchial glands are the seat of simple or tubercular inflammation.

The smaller bronchi may be dilated. In the portions of lung which are not consolidated the air spaces may be somewhat dilated. Occasionally some of the air spaces are ruptured and the septa between the lobules are infiltrated with air.

In order to understand the true anatomical characters of broncho-pneumonia, it is necessary to determine which of these different changes is essential and constant and which are accessory and inconstant.

The essential and constant lesion is a productive inflammation of the walls (not the mucous membrane) of the bronchi, and of the air spaces immediately surrounding the inflamed bronchi. The walls of the bronchi are thickened and infiltrated by a growth of new cells; the walls of the air spaces are thickened, their cavities are filled with new connective tissue or with fibrin, pus, and epithelium. The inflammation is from the first not exudative alone, but also productive, that is, with the formation of new tissue. It involves the medium-sized and smaller bronchi of both lungs, but is not everywhere equally severe; in some parts of the lungs the lesions are much more marked than in others.

The accessory lesions, some of which are present in one case and some in others, are:

1. A catarrhal inflammation of the mucous membrane of the bronchi.
2. An exudative inflammation of the air spaces, which fills their cavities with fibrin, pus, and epithelium, and produces consolidation of larger or smaller portions of the lungs. In young children the epithelial cells which line the air spaces are much more numerous than they are in adults, so when children's lungs are inflamed the epithelial cells form a larger part of the inflammatory product than they do in the lungs of adults.
3. An exudative inflammation of the pleura, which coats it with fibrin.
4. Dilatation of the bronchi, of which the walls are the seat of productive inflammation.
5. Areas of atelectasis.
6. Simple, or tubercular inflammation of the bronchial glands.

As the inflammation of the walls of the bronchi and of the air spaces surrounding them is from the first a productive inflammation, it follows the law which governs that form of inflammation. It is apt to last for a longer time than does an exudative inflammation, and it is liable to change into a chronic productive inflammation.

It is not uncommon, therefore, for a broncho-pneumonia to continue for several weeks, or to be followed by permanent changes in the lungs.

If the broncho-pneumonia becomes chronic the inflammation of the walls of the bronchi and of the air spaces which surround them continues; we then find that the bronchi are dilated, their walls are thickened, they are surrounded by zones of connective tissue; or part of a lobe, or an entire lobe, is entirely changed into connective tissue. The pulmonary pleura may also be very much thickened.

Symptoms.—In very young infants the only symptoms are: fever, prostration, and rapid breathing. There is no cough:

there are no physical signs. The disease is almost certainly fatal within a very few days.

In older children the broncho-pneumonia may be preceded by the symptoms of measles, of whooping-cough, of coryza, of pharyngitis, or of a catarrhal bronchitis of the larger tubes; or it may begin without having been preceded by any other morbid conditions.

There is a good deal of difference in the different cases as to the severity of the invasion. The more severe cases are ushered in by one or more general convulsions; or by a rapid rise of temperature, vomiting, oppressed breathing, and delirium. The milder cases begin with lower temperatures, moderate prostration, and increased frequency of breathing.

After the disease is established the patients continue to have a febrile movement. The temperature in most cases is irregular, but on many days up to 105° F. Very often the temperature is of distinctly remittent type, a morning temperature of 99° to 100° F., and an evening temperature of 104° to 105° F. It is to be noticed, however, that in children both bronchitis and lobar pneumonia are also often accompanied by fever of a remittent type. It may very well be that this disposition to remission belongs rather to the age of the patient than to the character of the disease. The height of the temperature varies from day to day, sometimes with the progress or extension of the inflammation, sometimes without discoverable cause. In the cases which recover it requires several days for the temperature to fall to the normal. But a sudden defervescence within twelve hours, like that of lobar pneumonia, is sometimes seen.

The height of the temperature is regularly in proportion to the severity of the broncho-pneumonia; with temperatures of over 105° F. the mortality of the disease is considerable. There are, however, patients in whom the temperature runs between 99° and 100° F. who do very badly.

In children the pulse is more rapid than it is in adults, and is also more easily rendered rapid by disease. So in broncho-pneumonia pulses of from 140 to 170 to the minute are not uncommon, and in bad cases the pulse can hardly be counted.

An increase in the frequency of the breathing is almost constantly present, as much as 40 to the minute even in mild cases, up to 60, 70, or 80 in the bad cases. It is of importance to notice not only the frequency of the breathing, but also how much air enters into the lungs. The breathing may be made worse for a time by distension of the stomach.

Sleeplessness, restlessness, and delirium are often present, and sometimes very troublesome. They seem to depend partly on the fever, partly on the interference with breathing, partly on the temperament of the child.

The face is flushed, the tongue is coated and sometimes dry, there may be vomiting or diarrhœa; sometimes there is pain over the chest.

Cough is often present, dependent on the catarrhal bronchitis rather than on the broncho-pneumonia. The sputa are swallowed rather than coughed up. They may collect in the stomach and be vomited up.

The urine may contain a little albumin and a few casts, the kidneys being the seat of acute degeneration.

The physical signs vary with the condition of the lung. If the inflammation is limited to the walls of the bronchi and to the air spaces immediately surrounding them, there are no physical signs. If a catarrhal bronchitis is present there are coarse and subcrepitant râles. If there is a diffuse pneumonia, with consolidation of a considerable portion of the lung, there are dulness on percussion and bronchial voice and breathing. If there is fibrin on the pleura, there are crepitant or subcrepitant râles. The signs of the consolidation and of the pleurisy are usually developed between the second and fifth days, but it is not uncommon for them to be delayed until a much later period.

The duration of broncho-pneumonia in children varies very much in different cases. Of the fatal cases the larger number die within two weeks, but some prove fatal within two days, and some are protracted for seven or eight weeks. In the cases which recover the constitutional symptoms continue for from one to three weeks in the majority of patients, but it is by no means unusual for the active symptoms to continue for six or eight weeks and yet the patients make a perfectly good recovery. Resolution requires a longer time than in lobar pneumonia, from seven to fourteen days in most of the cases.

CASE I.—Male, eight months old, April 16, 1891. Two days ago the child began to have malaise, a little cough and fever, but slept well and did not seem very ill.

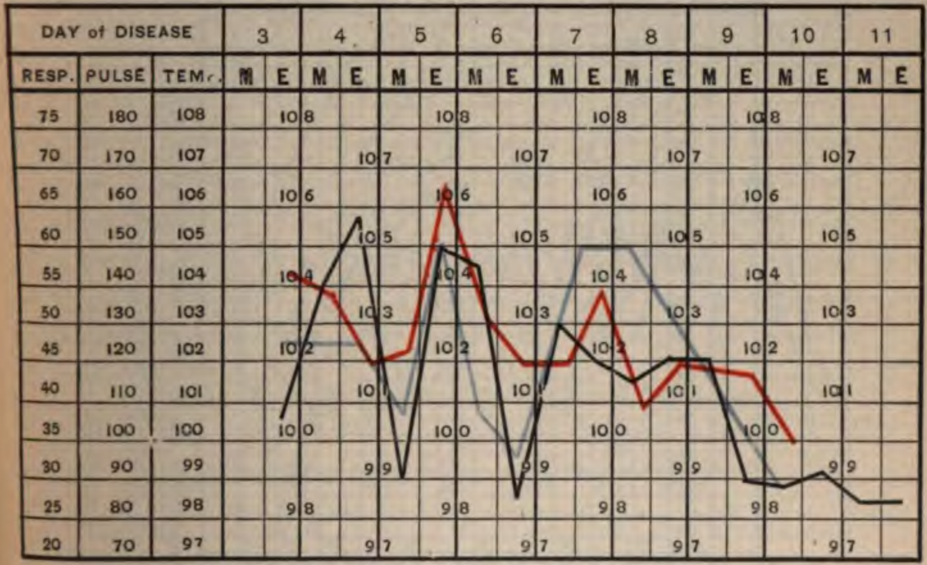
Now, on the third day, the child is well nourished; he is moderately sick; over the lower lobe of the left lung there are dulness on percussion and broncho-vesicular breathing.

On the fourth day he was given one-sixth of a grain of calomel every hour up to three doses, and a sponge bath.

On the tenth day he was well.

(For Chart of Case I, see Fig. 64.)

FIG. 64



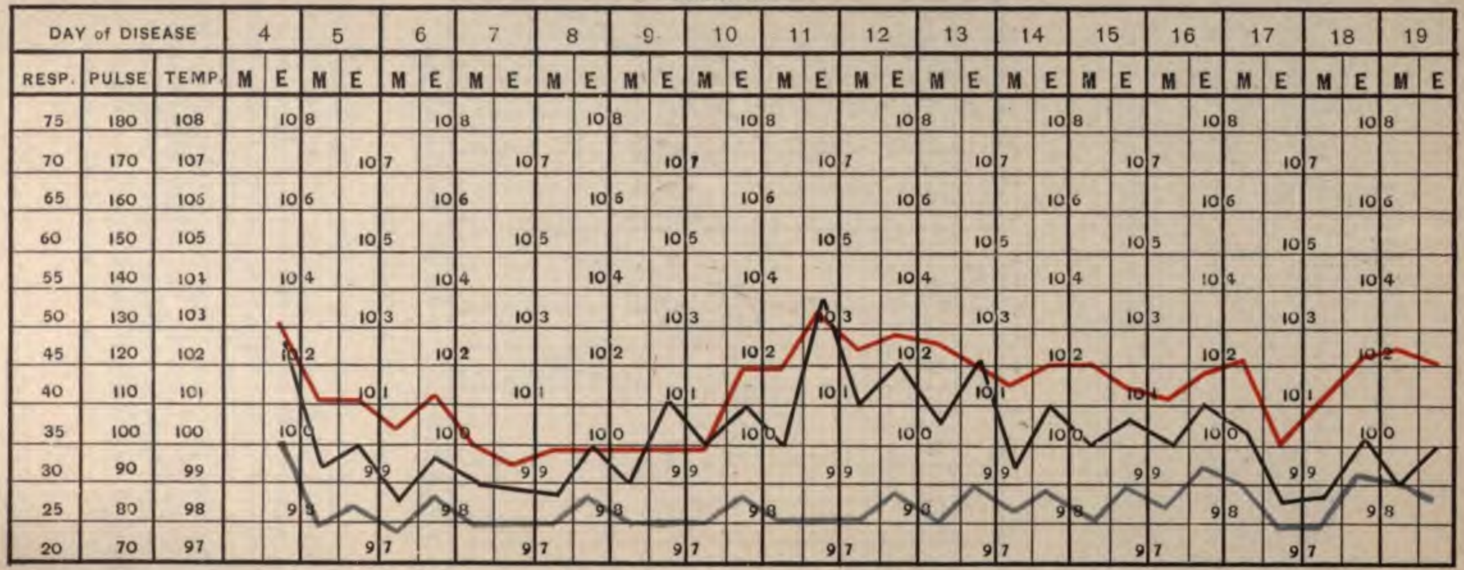
Temperature black, Pulse red, Respiration blue

CASE I.—BRONCHO-PNEUMONIA.

CASE II.—Male, 3 years old, January 28, 1898. The child was perfectly well until three days ago. Then he began to cough, with fever, pain in the side, vomiting, and diarrhœa. Now, on the fourth day, over the right lung behind in the midscapular region there is a small area of dulness with fine râles. Over the rest of both lungs there are sibilant breathing and coarse râles. The child gradually improved, the cough disappeared, and on the twenty-fourth day he was discharged cured.

(For Chart of Case II, see Fig. 65.)

FIG. 65



Temperature black, Pulse red, Respiration blue

CASE II.—BRONCHO-PNEUMONIA.

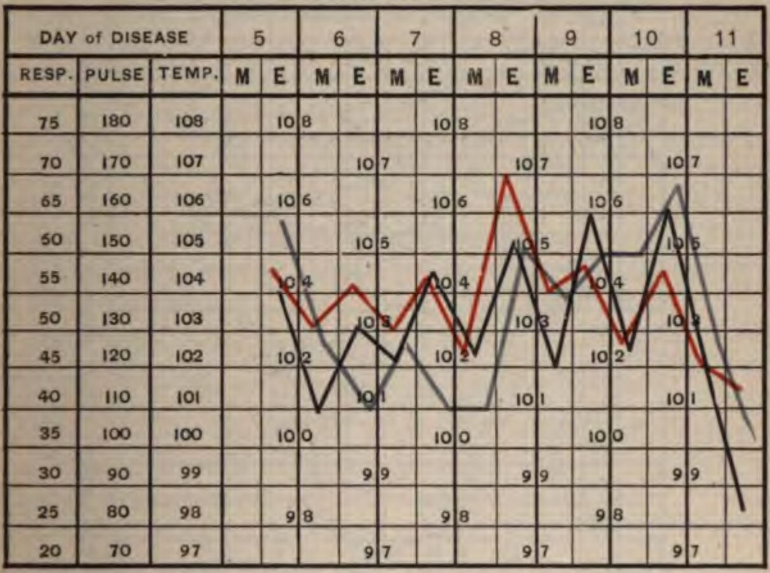
CASE III.—Male, 4 years old, November 26, 1896. Six days ago he began to cough. Four days ago he began to have pain in the right side, and fever with alternating delirium and stupor.

The child looked very ill and continued so till the eleventh day. There were crepitant and subcrepitant râles over both lungs, but no dulness on percussion. He received no treatment except a few drops of wine of ipecac.

On the seventeenth day he was discharged cured.

(For Chart of Case III, see Fig. 66.)

FIG. 66



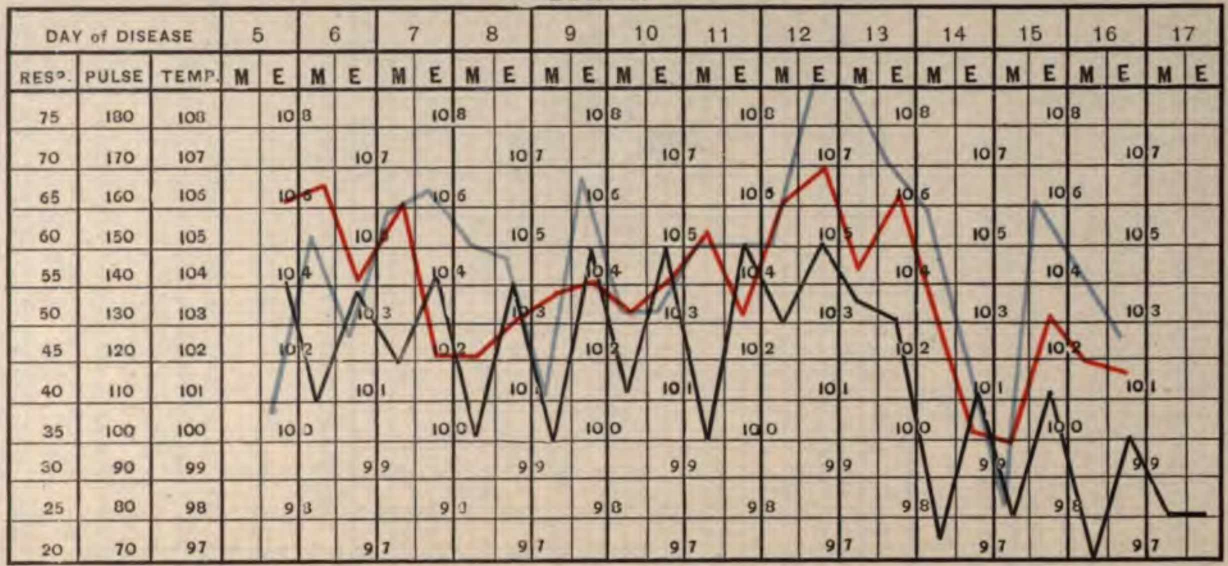
Temperature black, Pulse red, Respiration blue

CASE III.—BRONCHO-PNEUMONIA.

CASE IV.—Female, 3 years old, November 2, 1896. Four days ago, after having had a cough for several days, she became suddenly worse. She developed difficult breathing, cyanosis, and alternating stupor and delirium.

Now, on the fifth day, she is pale, the lips cyanotic, stupid and looks very ill. Over the right chest behind there are dulness on percussion, bronchial voice and breathing, crepitant and subcrepitant râles. She was given one-fourth of a grain of calomel every hour up to four doses. This was repeated the next day.

FIG. 67



Temperature black, Pulse red, Respiration blue

Case IV.—Broncho-Pneumonia.

As will be seen from the chart, she was very sick until the fourteenth day.

On the twenty-second day the physical signs had disappeared.

On the thirtieth day she was discharged cured.

(For Chart of Case IV, see Fig. 67.)

The Cerebral Cases.—In many of the cases of broncho-pneumonia there are cerebral symptoms—convulsions, restlessness, and delirium—but in some patients these symptoms are developed to such a degree, and are so out of proportion to the pulmonary symptoms, that the cases require a separate description.

The symptoms resemble those of an acute or a tubercular meningitis. They may begin and go on acutely, with high fever, prostration, one or more convulsions, alternating delirium and stupor. Or the course is more subacute—loss of appetite, vomiting, moderate prostration, not very high fever, alternating stupor and delirium. These symptoms may continue for from two to ten days before there are any pulmonary symptoms. Then, as the pulmonary symptoms are developed, the cerebral symptoms subside.

Persistent Cases.—If, after the subsidence of the acute broncho-pneumonia, a chronic inflammation persists, the children begin to improve, but yet do not get well.

In some the cough and the physical signs continue, the appetite is poor, the children do not gain flesh and strength, but yet they are not sick in bed—often not confined to the house.

In others the same symptoms exist, there is also an irregular fever, and the patients are sick in bed.

Of these protracted cases some recover entirely; some recover with permanent consolidation of a portion of the lungs; some die, exhausted by the disease; some go on to have a chronic interstitial pneumonia which lasts for many years.

In some cases of acute broncho-pneumonia the accompanying inflammation of the bronchial glands may be of tubercular character, and this may serve at a later period as the focus of infection which causes an acute general tuberculosis.

Treatment.—It must be confessed that there are still wide divergences of opinion as to the management and treatment of broncho-pneumonia in children.

It is taught by some that the children should be kept in a room with a temperature of 75° F. or over, the air of the room being kept moist by the constant production of steam.

On the other hand, many believe that high temperatures and moisture favor the growth of streptococci, deprive the child of good air, cause discomfort and restlessness, and favor the spreading of the inflammation.

Some believe that the heart action often fails, that this constitutes a serious feature of the disease, and that to avert this danger alcoholic stimulants, digitalis, strychnine, etc., are to be freely given.

Others think that in children under 5 years of age alcohol, digitalis, etc., make the heart's action worse instead of better, are likely to produce vomiting and diarrhœa, and increase the liability to death.

I believe that children have a good deal of capacity for recovering from broncho-pneumonia, and that high temperatures, rapid pulse and breathing, and delirium are not as serious symptoms in children as they are in adults. A temperature of 106° F., a pulse of 160, and breathing of 60 are seen often enough in children who make a good recovery.

Plenty of fresh air, good nursing and feeding, sometimes a little opium or aconite or acetanilid, seem to me to give the best results.

In adolescence the clinical picture of broncho-pneumonia is the same as in children, but the cerebral symptoms are not developed to the same extent, and the patients are more likely to cough up blood.

In adults the disease presents itself to us under several different forms:

1. The patient has an ordinary attack of catarrhal bronchitis lasting for several days. Instead of getting well promptly, however, the patients continue to cough and to feel sick, and on examining the chest we find a circumscribed area where there is dulness on percussion and loud, high-pitched voice. This consolidation of the lungs does not, however, last very long, and the patients make a good recovery.

2. The patients are suddenly attacked with a very severe and

general broncho-pneumonia. There are chills, a rapid rise of temperature, headache, pains in the back and chest, vomiting, great prostration, a rapid pulse which soon becomes feeble, very bad breathing—rapid, labored, and insufficient—venous congestion of the skin and of the viscera, cough, at first dry, then with profuse mucous and blood-stained sputa, sleeplessness, restlessness, and delirium, and albumin in the urine. There are coarse, subcrepitant and crepitant râles over both lungs, sibilant and sonorous breathing; the percussion note is normal or exaggerated or dull. The disease lasts for from seven to fourteen days; it is very apt to prove fatal.

Treatment.—The most efficient remedies are the energetic use of wet or dry cups over the entire chest, the administration of calomel or the sulphate of magnesia in small and repeated doses, ipecac, the inhalation of oxygen gas, and stimulants.

CASE V.—Male, 25 years old, December 19, 1881. Seven days ago he began to have cough, dyspnœa, headache, chills and fever. Now, on the eighth day, he coughs up large quantities of muco-purulent matter. He has great pain in the chest, urgent dyspnœa, and cyanosis. There is good resonance over both lungs, with coarse and subcrepitant râles. He continued in the same condition, with intense dyspnœa, cyanosis, profuse expectoration, delirium, temperature from 102° to 104°, until he died on the fourteenth day.

Autopsy.—The lungs are large and heavy; there is a little fibrin on the pulmonary pleura. All the bronchi contain muco-pus. The medium-sized and smaller bronchi show a cellular infiltration of their walls and are surrounded by zones of air vesicles filled with blood. The other viscera are normal except for venous congestion.

CASE VI. *Broncho-Pneumonia; Death.*—Male, 29 years old, October 25, 1896. About a week ago he had a cold for three days, but did not go to bed. He had cough with mucous expectoration, headache, and pains in the chest and back. For the past two days the cough and pain have been worse and he has had continuous dyspnœa.

Now, on the third day of the broncho-pneumonia, the temperature is 101° F., pulse 120, breathing 24; urine 1022, no albumin, hyaline and granular casts. The face and fingers are very cyanotic; the breathing is labored; the arteries are a little contracted.

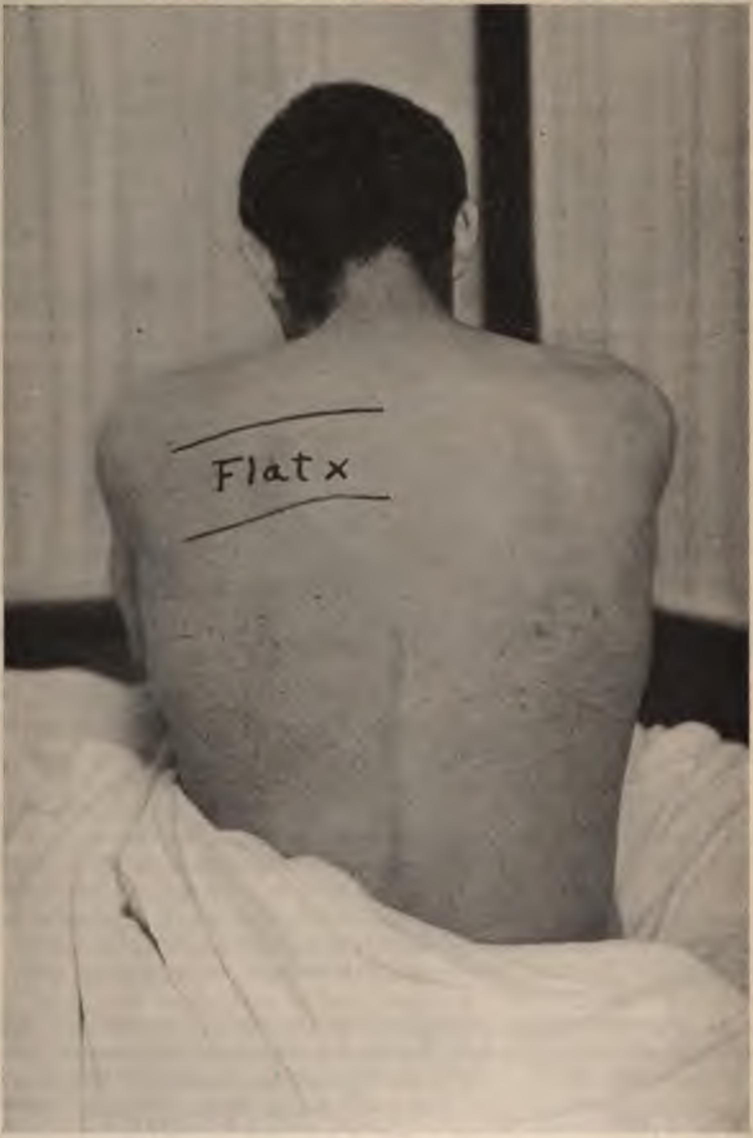


FIG. 68.

The man went on with morning temperatures of about 100° F. and evening temperatures of about 103° F. until January 8, the sixty-second day of his disease; from then till the seventy-eighth day the temperature was not over 100° F., after that it was normal.

He continued to have an abundant muco-purulent sputum until the fifty-second day, when the quantity began to diminish, and gradually became less and less until it disappeared. The sputum was repeatedly examined; it did not contain tubercle bacilli, actinomyces, or lung tissue.

On the twenty-sixth day there was dulness and a friction sound over the lower half of the left lung; on the forty-third day, over the middle of the left lung behind, flatness and absence of breathing, and below this, feeble breathing and subcrepitant râles; on the fifty-third day, over the area of flatness, broncho-vesicular breathing and subcrepitant râles were heard. On the eighty-fourth day the flatness had disappeared, the breathing was broncho-vesicular with an occasional friction sound.

On the forty-third day a large aspirator needle was put in deeply at the point of greatest flatness (marked X on the photograph, Fig. 68); it drew only blood.

On the eighty-sixth day the man went home apparently well.

(For Chart of Case VIII, see Fig. 69.)

Case IX.—Male, 19 years old, February 5, 1883. Three weeks ago he began to have cough with profuse expectoration, fever, and night sweats.

Now, on the twenty-second day, he has cough with profuse expectoration, his tongue is brown and dry, and he is delirious. He continued in this condition until he died on the forty-sixth day.

Autopsy.—The right pleural cavity is half full of turbid serum; the pleura is coated with fibrin.

The left pulmonary pleura is coated with fibrin.

The right upper lobe is in the condition of red hepatization mottled with white spots. The lower lobe is compressed and dotted with pea-sized white nodules. In the diffuse hepatization the air spaces are filled with fibrin and pus.

The white nodules are small bronchi surrounded by zones of hepatization. The cavities of these bronchi contain pus; in some of them the epithelium is in place. The walls of the bronchi are infiltrated with cells, some to such an extent that the wall of the bronchus is destroyed. Many of these bronchi are dilated so as to form small cavities.

Around the bronchi the zones of hepatization are made up of air vesicles, of which the walls are thickened and infiltrated with cells. Their cavities are filled with pus cells, epithelial cells, small round, polygonal, and fusiform cells embedded in a basement substance in places like fibrin, in places homogeneous.

(For Chart of Case IX, see Fig. 70.)

Treatment.—The patients are to be kept in bed and as well fed as possible. The most efficacious treatment seems to be the continued inhalation of the vapor of creosote.

5. Persons suffering from emphysema sometimes get up a subacute broncho-pneumonia, which may prove fatal.

CASE X.—A male, 60 years old, was in the hospital for several months with emphysema and bronchitis. He was well enough to be up and walking about until twenty-four hours before his death. Then he suddenly became worse and died.

Autopsy.—The brain was not examined. There are extensive old pleuritic adhesions over both lungs. Both lungs are emphysematous. The left lung is not congested or œdematous.

The apex of the right lung is infiltrated with bloody serum. The lower lobe is in the condition of partial diffuse, gray, gelatinous hepatization mottled with small yellow spots. The diffuse hepatization consists of air spaces filled with fibrin and a few large cells. The walls of the small bronchi are thickened and infiltrated with cells, but the epithelium is unchanged. The yellow spots are air spaces filled with pus.

The larynx and trachea are normal.

The heart shows a little old thickening of the aortic valves.

The liver is a nutmeg liver.

The kidneys show the changes of chronic nephritis with atrophy.

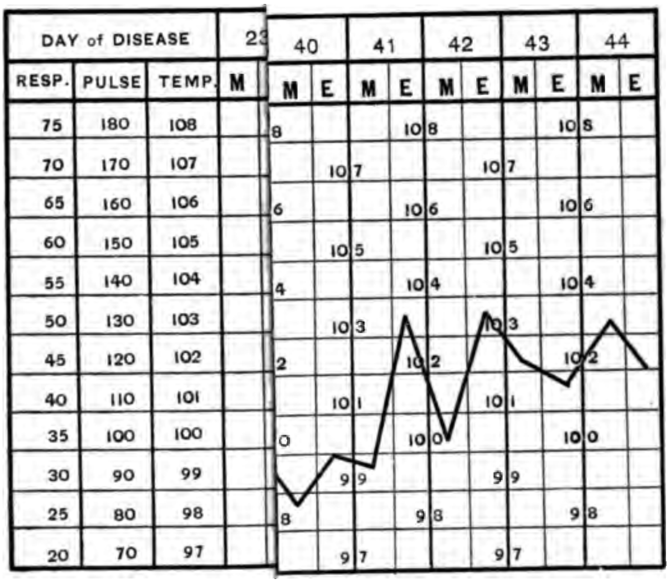
The stomach is in the condition of chronic gastritis.

The spleen is normal.

6. Broncho-pneumonia, especially of the lower lobes, is secondary to the infectious diseases, to injuries, to surgical operations, and to many conditions which are likely to cause congestion of the lungs and the inhalation of streptococci.

THE PNEUMONIA OF HEART DISEASE.

Definition.—A chronic productive inflammation of the lungs caused by chronic congestion, and resulting in thickening of the


DAY of DISEASE
23
40
41
42
43
44
RESP.
PULSE
TEMP.
M
M E M E M E M E M E
75 180 108
70 170 107
65 160 106
60 150 105
55 140 104
50 130 103
45 120 102
40 110 101
35 100 100
30 90 99
25 80 98
20 70 97
8
6
4
2
0
8
10 8
10 7
10 6
10 5
10 4
10 3
10 2
10 1
10 0
9 9
9 8
9 7

walls of the air spaces, filling of their cavities with epithelial cells, and a deposition of pigment.

Synonyms.—Brown, or pigment, induration of the lungs.

Etiology.—Any long-continued mechanical obstruction to the escape of the blood from the lungs into the left cavities of the heart can produce this form of pneumonia. The most frequent and effectual obstruction is furnished by a stenosis of the mitral valves; but any valvular lesion of the heart, dilatation of the ventricles, or aneurism of the arch of the aorta may act in the same way.

Morbid Anatomy.—The first effect of the obstruction to the circulation seems to be a change in the capillary vessels in the walls of the air spaces. These vessels become dilated, tortuous, and have their walls thickened. Then there is a gradual thickening and pigmentation of the walls of the air spaces; an increase in the size and number of the epithelial cells, until they partly or completely fill the cavities of the air spaces; an escape of the red blood cells into the air spaces. Finally, when the inflammation has reached its full development, there is a smooth, red hepatization of portions of both lungs, an hepatization due principally to the filling of the air spaces with epithelial cells.

The lungs are diminished in size, sometimes covered with old adhesions, but seldom with fibrin. There may be more or less serum of dropsical character in the pleural cavities. The texture of the lungs is leathery and dense, or that of a smooth hepatization. They are dry, of a salmon-pink color mottled with brown or black. There may be large or small areas where the air spaces are filled with extravasated blood.

Symptoms.—The principal symptoms exhibited by the patients are those due to the lesions of the heart or aorta. The changes in the lungs do not give as marked symptoms as might be expected. The physical signs are obscure—more or less dulness and diminished breathing. The rational symptoms are—dyspnœa, cough, mucous and bloody sputa, the continued expectoration of pure blood.

Treatment.—It is evident that this condition of the lungs is one which cannot be influenced by treatment. We simply attend as well as we can to the disturbances of circulation which have caused the lung trouble.

INTERSTITIAL PNEUMONIA.

Definition.—A chronic productive inflammation which involves the connective-tissue framework of the lungs and the walls of the air spaces, and results in the formation of new connective tissue and obliteration of the air spaces.

Etiology.—The well-marked examples of interstitial pneumonia follow acute lobar pneumonia with the production of new connective tissue; broncho-pneumonia; chronic pleurisy with adhesions; chronic bronchitis; and the inhalation of the dust of coal or of stone.

Morbid Anatomy.—The condition of the lungs varies with the cause of the interstitial pneumonia.

1. If it follows acute lobar pneumonia with the production of new connective tissue, one lobe, or an entire lung, is covered with pleuritic adhesions; it is small, smooth, and dense; the air spaces and small bronchi are obliterated by the new connective tissue.

2. If it follows broncho-pneumonia, one or more lobes are studded with fibrous nodules, or are converted into dense fibrous tissue. The pleura is thickened, the bronchi are inflamed and often dilated.

3. If it follows thickening of the pleura, bands of connective tissue extend from the pleura into the lung, the bronchi are inflamed and often dilated.

4. If it follows chronic bronchitis there are fibrous nodules around the bronchi, with more or less diffuse connective tissue.

5. If it is due to the inhalation of the dust of coal or stone, we find in both lungs fibrous peribronchitic nodules and diffuse connective tissue.

In most of the cases the portions of lung exempt from the interstitial pneumonia are emphysematous.

Symptoms.—The patients have a cough with mucous expectoration. The cough becomes more constant and troublesome as the disease progresses. The expectoration becomes muco-purulent, sometimes fetid. If the bronchi become dilated, the muco-pus accumulates in them and is coughed up at intervals in large quantities. There may be occasional hæmoptyses. There is dyspnœa on exertion, at first slight, later more marked. There may be uncomfortable feelings or actual pain over the affected side of the chest. There is gradual loss of flesh and of strength. Neither laryngitis nor diarrhœa belongs to the disease. There is no fever except with exacerbations of the bronchitis.

The affected side of the chest is retracted, the other side is enlarged, the heart is displaced, the vertebral column is curved laterally. There is, on percussion, dulness or flatness or tympanitic resonance. The vocal fremitus is increased. The breathing is sonorous, sibilant, bronchial, cavernous, or amphoric, according to the condition of the bronchi and the degree of consolidation of the lung. There are pleuritic creaking sounds, subcrepitant, coarse, or gurgling râles.

The disease is one which lasts for many years, and the patients usually die with some acute inflammation of the other healthy lung.

The cases which we see most frequently are those which have followed a broncho-pneumonia occurring in childhood. The history will be that the patient has had an attack of inflammation of the lungs when a child; that ever since then he has had a cough, sometimes better, sometimes worse, but with the quantity of muco-purulent sputum gradually increasing; that he has had several other attacks of inflammation of the lungs, and yet after this long history of cough and expectoration, and with the physical signs of a lobe which is small, consolidated, and contains dilated bronchi, it will be found that the patient is well nourished and capable of considerable mental and physical exertion.

Treatment.—The most efficient treatment is that the patient should reside permanently in a climate where he is able to live out of doors and where his bronchitis does well.

The drug treatment is that of a chronic bronchitis.

TUBERCULAR PNEUMONIA.

We include under this name all the inflammations of the lungs which are caused by the presence of tubercle bacilli in these organs.

Tubercle bacilli in any part of the body, by their own irritating properties and those of their toxins, may cause: exudation from the blood vessels; a growth of round-celled tissue, tubercle tissue, or fibrous tissue; or necrosis of tissue. These inflammatory changes may occur separately or together.

In the lung the particular form of inflammation excited seems to depend upon the number of tubercle bacilli and the way in which they are introduced into the lungs.

If large numbers of tubercle bacilli are inhaled and find their way into the bronchi and air spaces, a considerable part of the lung becomes the seat of exudative and productive inflammation.

If smaller numbers of these bacilli are inhaled, or are brought into the lungs by the blood vessels or lymphatics, there is little or no exudative inflammation, and the productive inflammation results in the formation of miliary tubercles.

There is, in addition, in a certain number of cases, an infection of the lung by streptococci or pneumococci, which are additional irritants, produce exudation, and increase the liability to necrosis and suppuration.

There are, therefore, a number of anatomical forms of tubercular pneumonia. For these we have as yet no good names, so that we continue to use the old arbitrary designations.

We distinguish, therefore:

Acute miliary tuberculosis of the lungs.
Subacute miliary tuberculosis of the lungs.
Chronic miliary tuberculosis of the lungs.
Acute pulmonary phthisis.
Chronic pulmonary phthisis.

ACUTE MILIARY TUBERCULOSIS OF THE LUNGS.

Definition.—A tubercular inflammation of the lungs attended with the formation of numerous small inflammatory foci, called miliary tubercles.

Etiology.—For the development of tubercular inflammation in any part of the body there are necessary: the proper predisposition of the individual, a local cause of inflammation, and the growth of tubercle bacilli. In the lungs the tubercle bacilli seem to be either inhaled or brought to the lungs by the blood. It is possible that the bacilli may be first inhaled, then collected in the bronchial glands, and from the glands find their way into the pulmonary circulation. Certainly, we see cases in which tubercular inflammation of the bronchial glands precedes miliary tuberculosis of the lungs. The bacilli which are inhaled must be derived to some extent from the dried sputa of persons who have tubercular pneumonia. The bacilli are much more abundant in the air of some localities than of others, and at high altitudes and on the ocean the air contains none of these organisms. The bacilli which are conveyed to the lungs by the blood are derived from a focus of tubercular inflammation in some other parts of the body. In man tuberculosis is conveyed by inoculation from one person to another only in rare cases. In some animals tuberculosis can be inoculated at will, and miliary tubercles of the lungs easily produced. Trudeau, however, has shown that the success of such an inoculation can be influenced by environment, and that it is possible to keep rabbits under such conditions of good health that they cease to offer a good soil for the growth of tubercle bacilli. The most perfect miliary tubercles in the lungs of animals which I have ever seen are those produced by injections of dead tubercle bacilli into the trachea of rabbits (Prudden).

Morbid Anatomy.—The miliary tubercles may be confined to part of a lung, or be distributed through both lungs. They are close together, or separated by considerable intervals, or aggregated together into larger masses. They are of gray, white, or yellow color. They all contain tubercle bacilli, but these are much more numerous in some tubercles than they are in others. They are composed of:

1. Groups of air passages and air vesicles filled with granular matter, pus cells, and epithelium.
2. Groups of air passages and air vesicles of which the walls are infiltrated with tubercle tissue, while their cavities are filled either with tubercle tissue or with epithelium, pus, and fibrin.
3. Infiltrations of the walls of small bronchi with tubercle tissue or round-celled tissue, the infiltration extending to the walls of the adjacent air spaces.
4. Nodules of tubercle tissue situated in the pulmonary pleura,

the septa between the lobules, the walls of the bronchi, and the walls of the veins.

In addition to the presence of the miliary tubercles the bronchi are congested and coated with mucus, the walls of the air spaces are congested, the epithelial cells which line them are increased in size and number, some of the air spaces are filled with epithelium, fibrin, and pus; there may be fibrin on the pulmonary pleura.

Symptoms.—The method of infection is such that an enormous number of miliary tubercles are formed in the lungs and in other parts of the body, the poisonous effects of the chemical substances produced by the growth of the tubercle bacilli are very marked, and the patients behave as if they had a general disease rather than an inflammation of the lungs.

Although the lungs are congested and thickly studded with miliary tubercles, the physical signs are not as constant or as plain as one would wish them to be. The changes in the percussion note are not well marked. There may or may not be some dulness. Crepitant or subcrepitant or coarse râles, bronchial voice and breathing, or rude breathing are sometimes present, sometimes absent. Moreover, the physical signs may be modified by the existence of old tubercular lesions in the lungs. The rational pulmonary symptoms also are not constant. Cough and mucous sputa, rapid and oppressed breathing, and pain over the chest are present in some cases, absent in others.

The symptoms of the acute tuberculosis come on suddenly or gradually, or are preceded by those of tubercular inflammation of the lungs or of some other part of the body. The symptoms of some of the cases have already been described under the head of tubercular meningitis. The cases can be roughly grouped into:

Cases with marked cerebral symptoms.

Cases with pulmonary symptoms.

Cases with only the symptoms of general infection.

Obscure cases with the symptoms masked by those of other lesions.

The cerebral cases have already been described with tubercular meningitis.

In the cases with pulmonary symptoms, the patients regularly have cough with muco-purulent sputum in which tubercle bacilli are found. As a rule there are no hæmoptyses unless the general tuberculosis follows a previous localized pulmonary tuberculosis.

The physical signs are uncertain. They depend upon the presence of accompanying dry pleurisy, bronchitis, imperfect consolidation, or old tubercular lesions.

The breathing is regularly rapid and difficult. This may be a marked symptom.

The pulse becomes more and more rapid as the disease goes on.

The temperature is very variable and often not in proportion to the severity of the disease. It is regularly higher in the afternoon.

At some time in the disease stupor and delirium are developed.

The invasion of the symptoms is acute or subacute.

The disease lasts from a few days to several months and is regularly fatal.

CASE I. *Acute Miliary Tuberculosis, about six weeks' duration.*—Male, 24 years old. Six weeks before death he caught cold and had pain over his chest, dry and hacking cough, night sweats, and rapidly lost flesh and strength. In the fifth week there were dulness and increased vocal fremitus over both apices, slight dulness over both lower lobes, rude respiration, and subcrepitant râles all over both lungs.

The temperature ran from 100° to 102° F., the pulse from 92 to 140, the breathing from 64 to 68. The dyspnœa was most urgent till he died.

Autopsy.—The membranes of the brain are normal.

The heart, stomach, intestines, and peritoneum are normal.

The liver, spleen, and kidneys are studded with small gray tubercles.

In the lungs there is muco-pus in the bronchi. Both lungs are thickly studded with small gray tubercles. In the upper lobes the tubercles are larger, white, and opaque, and there is a little red hepatization between them.

CASE II. *Acute Miliary Tuberculosis, Pulmonary Symptoms.*—Female, 22 years old, April 8, 1874. Three months ago she caught cold and was quite sick for several days with cough and fever. From this she recovered, but the cough with a little expectoration has continued ever since.

Two weeks ago she suddenly became worse. The cough was more frequent, with rusty sputum, and she went to bed. Now the temperature is 103° F., pulse 150, breathing 56. There are dulness on percussion, sibilant and sonorous breathing, with coarse and subcrepitant râles over both lungs.

The temperature continued between 101° and 105° F., the pulse 120 to 150, the breathing 40 to 50. She grew steadily worse, with dry tongue, increasing dyspnœa, and delirium, until a week later, when she died.

Autopsy.—The brain and its membranes are normal.

The heart is normal.

The pulmonary pleuræ of both lungs are studded with miliary tubercles.

The mucous membrane of the bronchi is congested. Both lungs are thickly studded with gray miliary tubercles.

The liver, the spleen, the kidneys, and the diaphragm contain miliary tubercles.

In the cases with the symptoms of general infection the marked features are the development of the typhoid state with fever, rapid emaciation, delirium, and stupor. The disease very closely resembles typhoid fever or malignant endocarditis.

The invasion is acute or subacute, or follows a subacute or chronic miliary tuberculosis.

The disease only lasts a few days, or is protracted over many weeks.

CASE III. *Acute Miliary Tuberculosis of seven days' duration.*—Male, 77 years old, November 3, 1874. Four days ago he was attacked with chills, fever, and great prostration. He had no pain, headache, vomiting, diarrhœa, or cough.

Now, on the fifth day, he is very weak, temperature 102° F., pulse 88, slight delirium, no cough, no difficulty in breathing, no physical signs over the lungs.

He continued in the same condition until the seventh day, when he died.

Autopsy.—There is a considerable quantity of clear serum beneath the pia mater.

The heart is normal; the arch of the aorta is dilated and atheromatous. There are extensive old pleuritic adhesions over both lungs. Both lungs are thickly studded with miliary tubercles, and many of the air spaces contain epithelial cells and fibrin.

The liver and spleen are normal.

The kidneys contain a few miliary tubercles.

CASE IV. *Acute Miliary Tuberculosis, subacute invasion.*—Male, 58 years old, July 1, 1880. Six weeks ago he began to have an irregular fever and lost flesh. Three weeks ago he began to have dyspnœa on exertion, and pain in the right side.

Now, in the seventh week, he is feeble and emaciated; some dyspnœa; no physical signs over the lungs; urine 1023, no albumin. The temperature ran from 99° to 102° F., pulse 88-104, breathing 22-44. He passed into the typhoid state and died in the eighth week.

Autopsy.—The heart is normal.

There is an old fibrous nodule at the apex of the right lung; there are no evidences of bronchitis; both lungs are thickly studded with miliary tubercles.

The liver weighs four pounds and is cirrhotic.

The spleen is normal.

The kidneys are normal.

The left seminal vesicle and the right epididymis are the seat of chronic tubercular inflammation.

The cases in which the symptoms of some other disease are prominent are often so obscure that the diagnosis is hardly possible.

CASE V. *Acute Miliary Tuberculosis with the Symptoms of Cirrhosis of the Liver and Jaundice.*—Male, 41 years old, October 14, 1876. Five months ago he began to have œdema of the feet.

For two weeks he has had ascites and jaundice.

Now he is fairly nourished; the pulse and temperature are normal. He is jaundiced and has ascites; urine 1014, bile pigment, a trace of albumin.

October 18: The jaundice and ascites were increasing and he had vomiting and diarrhœa.

October 25 he died.

Autopsy.—The dura mater and the pia mater are thickened.

The heart is normal.

The left pleura is thickened and adherent.

The right pleural cavity contains serum.

Both lungs are studded with large, yellow miliary tubercles.

The spleen contains large, yellow tubercles.

The liver is of medium size, cirrhotic, and contains small miliary tubercles.

The stomach is the seat of chronic catarrhal gastritis.

The duodenum is the seat of acute catarrhal inflammation extending into the common bile duct.

The kidneys are the seat of chronic nephritis and contain miliary tubercles.

The peritoneal cavity is distended with serum.

SUBACUTE MILIARY TUBERCULOSIS OF THE LUNGS.

This is altogether the most frequent form of pulmonary tuberculosis. The picture is that of a localized tuberculosis with infections of different degrees of virulence and the association of other pulmonary inflammations.

Etiology.—This condition may be developed at any time in a person who has the tubercular disposition, and we are seldom certain in such cases as to the exciting cause of the inflammation or the exact method of the infection. It is also of frequent occurrence in persons who already have a chronic tubercular inflammation of some part of the lung. In such cases it is probable that the infection comes from the old tubercular lesion.

Morbid Anatomy.—As the disease runs rather a subacute than an acute course, the tubercles are firmer and more regularly composed of tubercle tissue than is the case in acute general tuberculosis. The most frequent position of the tubercles is at the apex of one lung, but they may be localized at any part of the lungs. It is not often that the whole of both lungs is involved. The cases vary greatly as to the extent of the associated bronchitis, pleurisy, and exudative pneumonia. The bronchitis may be confined to the portion of the lung which contains the miliary tubercles, or it may be a general bronchitis involving the larger tubes in both lungs. The pleurisy is either with fibrin alone, or with large quantities of serum in the pleural cavity. The exudative pneumonia may involve only small portions of the lung, or an entire lobe may be consolidated.

The cases of subacute miliary tuberculosis of the lungs may be conveniently grouped as follows:

1. Patients with well-marked physical signs and constitutional symptoms of infection. This means that in the affected portion of the lung, besides the miliary tubercles, there are also present dry pleurisy, bronchitis, and exudation into the air vesicles; also that the growth of the tubercle bacilli is attended with a considerable formation of toxin.

In some of the patients the invasion is gradual, with a little cough, small hæmoptyses, an afternoon fever, a pleurisy, or gradual loss of flesh and strength. But there are persons in whom the initial hæmoptysis comes on while they are apparently in robust health.

The ordinary position of the tubercular inflammation is at the apex of one or both lungs, but no part of the lungs is exempt.

The physical signs are those of a small, imperfect consolidation, localized bronchitis, and dry pleurisy. A change in the quality of the percussion note, sometimes amounting to dulness; a change in the quality of the breathing; friction sounds; subcrepitant râles, and increased vocal fremitus, are the regular physical signs.

The cough is in the morning, or throughout the day, or in attacks. Attacks of cough which produce vomiting, excluding whooping cough, regularly indicate pulmonary tuberculosis. With tubercular laryngitis the cough is much more distressing.

The sputum is muco-purulent, and tubercle bacilli should be found in the purulent portions.

Small hæmoptyses often mark the invasion of the disease and may be continued throughout its course.

Increased frequency of the pulse and of the breathing are often present.

The functions of the stomach are often disturbed, with loss of appetite, nausea and vomiting.

There is a rise of temperature, regularly higher in the afternoon and nearly normal in the morning.

The patients lose flesh and strength to a very considerable degree.

2. Patients with physical signs, but with no constitutional symptoms. In these patients the physical signs, the cough, the sputum, are the same as in the first set of cases. But there is no afternoon fever and very little loss of nutrition.

3. Patients with constitutional symptoms, but no physical signs. This is a large and troublesome class of cases. Errors in diagnosis are frequent and sometimes unavoidable. Patients who do not have tuberculosis are said to have it, and in persons who really have it the diagnosis is not made. We distinguish:

(*a*) Cases with loss of flesh and strength, with more or less gastric symptoms. There is nothing diagnostic in the blood of these patients. For a time there is no cough and no sputum. The cases that most closely resemble these patients are those of gastric dilatation and emaciation with rapid heart action but no gastric symptoms, and those of unaccountable emaciation in young women.

(*b*) Cases with persistent cough, with or without expectoration. If there are tubercle bacilli in the sputum, or if the cough causes vomiting, the diagnosis of tuberculosis is probable. But there are many other cases of persistent cough which cause much anxiety. Chronic pharyngitis, subacute laryngitis, bronchitis without physical signs, may give a cough for months. A true hysterical cough is easily recognized, but the women who have an ordinary persistent cough, that lasts for months in spite of all remedies, give one a great deal of trouble.

(*c*) Cases in which increased frequency of the pulse is for a time the only symptom. From these we have to distinguish the obscure cases of tachycardia.

(*d*) Cases in which increased frequency of breathing is for a time the only symptom. These patients must not be confounded with cases of hysteria and anæmia.

(*e*) Cases in which afternoon fever is the only symptom. These cases are to me the most troublesome of all, for the fever may last for months before pulmonary symptoms are developed, and other causes of afternoon fever are numerous and in themselves obscure.

Malignant endocarditis without cardiac signs or disturbances of circulation; chronic sepsis without a discoverable focus; the obscure and long-continued fevers of New York; syphilis and anæmia—each of these can give an afternoon fever that lasts for months.

√ Most curious of all are the young women who have an afternoon fever for months without any evident reason for it.

(*f*) Cases in which repeated hæmoptyses are for a time the only symptom. If these patients are really tubercular, other symptoms are usually developed within a few weeks.

The non-tubercular hæmoptyses are described later on.

4. Cases with associated inflammations of the lung.

The bronchitis may be general, with increased cough, dyspnœa, and fever. The exudation into the air spaces may be sufficient to consolidate portions of the lung of some little size.

The pleurisy, instead of being a dry pleurisy, may be one with a large effusion of serum.

Course of the Disease.—As the disease goes on some of the patients continue to have afternoon fever, a rapid heart action, and loss of nutrition, as the only symptoms. These are the simplest cases, the symptoms apparently depending entirely on poisoning by the toxin of the tubercle bacillus. Of these patients some are sick for several months and then recover altogether. In some the tubercular inflammation remains localized, but becomes chronic, and the patients go on to have the history of chronic miliary tuberculosis.

Instead of this, the tubercular inflammation, after remaining for months circumscribed, will either continuously or at intervals extend and involve more and more of the lungs. When this is the case the patients get worse either continuously or with intervals of improvement.

When, besides the miliary tubercles, pleurisy, bronchitis, or pneumonia is added, the cases are no longer simple, for these additional inflammations may be caused by the tubercle bacilli, by streptococci, by pneumococci, by the bacilli of influenza, and by other cases of inflammation. It can hardly be a matter of indifference as to whether these associated inflammations are produced by one or another of these causes, and yet this is a point which it is difficult to determine.

There are cases in which a pleurisy with effusion, or a general bronchitis, or a lobar pneumonia occurs in a person already having a subacute miliary tuberculosis. This may cause death, and after death no tubercle bacilli will be found except in the miliary tubercles. Or any one of these inflammations may run its course, the patient recover, and there will be no change in the course of the tuberculosis. Or the patient recovers from the accessory inflammation, but this seems to be a starting point for an extension of the tubercular inflammation.

On the other hand, there are cases in which the pleurisy, or bronchitis, or pneumonia is circumscribed. It is confined to the portions of the lung where the tubercles are situated, and it seems as if it were probably caused by the irritation of the toxins of the tubercle bacilli.

At the commencement of a case of subacute miliary tuberculosis with the lesions confined to one apex, it is impossible to predict the future of the patient. A fair proportion of the cases, under favorable conditions, recover, but there are many patients in whom the miliary tuberculosis and the accessory inflammations continue to extend.

The worst cases of all are those in which the whole of one lung, or a large part of both lungs, is from the first occupied by large numbers of miliary tubercles. These patients lose flesh and strength rapidly, have high afternoon temperatures, develop alternating stupor and delirium, pass into the typhoid condition, and die within a few weeks.

Treatment.—In the management of subacute miliary tuberculosis it is necessary to determine whether the patients can be sent to a suitable climate or whether they are obliged to stay at home.

In the choice of a suitable climate it is necessary to remember that a considerable number of cases of subacute miliary tuberculosis can get entirely well, but that this implies a residence of one or two years, or sometimes residence for life, in a suitable climate. This means that we must think of occupations and amusements for the patient. Evidently no one place is suitable for all the patients. Some do best on the seashore, some in the interior; some in a warm climate, some in a cold climate; while some do best if they travel from place to place.

If the patients have to remain at home we must distinguish between those who have an afternoon fever and loss of nutrition and those who have no fever and are fairly well nourished.

Those who have no fever can be allowed to go out of doors when the sun shines. But those who have much fever must remain in the house during the cold and wet months of the year. In such patients massage and passive motion are needed to take the place of exercise. In some of the patients a complete loss of appetite, or even dislike of food, is present, and has a large share in the loss of nutrition. The feeding of these patients is very difficult, and sometimes can only be effected with the stomach tube.

As regards medicinal treatment, the only drugs which have stood the test of time are: creosote, the carbonate of creosote, guaiacol and the carbonate of guaiacol. These are given by the stomach, the rectum, and by inhalation. While in no sense specifics, a certain number of patients improve under the use of these drugs.

It seems hardly necessary to enumerate the cures for tuberculosis which are constantly announced and forgotten. No one of them has yet stood the test of time and numbers.

The cough may depend upon irritation of the pharynx, larynx, bronchi, or pleura. So far as possible it should be treated by local applications, but there are patients who cannot be relieved except by some preparation of opium.

The distinction between subacute miliary tuberculosis and chronic miliary tuberculosis is to some extent an arbitrary one. But yet there is a real difference in their behavior. This seems to depend a good deal on the character of the associated inflammations.

In subacute miliary tuberculosis the bronchitis, dry pleurisy, and exudative pneumonia are transitory lesions which may disappear and leave only the miliary tubercles in the lungs.

In chronic miliary tuberculosis the bronchitis is more persistent. The interstitial pneumonia, bronchiectasiæ, and connective-tissue pleuritic adhesions are permanent lesions.

The virulence of the tubercular infection varies very much in both these forms of tuberculosis. In either one it may be mild or severe.

It may be said that in subacute tuberculosis the course is subacute with exacerbations.

In chronic tuberculosis the course is chronic, the patients getting worse as more tubercles are formed in the lungs.

Chronic Miliary Tuberculosis.

Definition.—A chronic tubercular inflammation of the lungs characterized by the formation of miliary tubercles, to which may be added bronchitis, dilatation of the bronchi, pleurisy, interstitial pneumonia, and emphysema.

Etiology.—It seems to be necessary to suppose in these cases a predisposition, an exciting cause for inflammation, and the growth of tubercle bacilli. It is evident, however, that the mode and character of the infection must differ from those of acute miliary tuberculosis. The structure of the tubercles is different, they contain very few tubercle bacilli, and the other morbid changes in the lungs may be of more importance than are the tubercles.

Morbid Anatomy.—The morbid process begins regularly at the apex of one of the lungs, and then slowly extends, either progressively or in attacks, until a larger portion of the lungs is involved.

In the simplest form of the disease the only change in the lungs is the formation of miliary tubercles. These tubercles are harder and denser than are those which are found with acute tuberculosis. They are composed of tubercle tissue, or round-celled tissue, or connective tissue, or are in the condition of cheesy degeneration. They contain but few tubercle bacilli.

Another simple form of the disease is when miliary tubercles alone are formed in lungs which are already the seat of vesicular emphysema.

Usually, however, in addition to the miliary tubercles there are other changes in the lungs. These additional lesions begin in the same part of the lung where the tubercles are formed, and accompany the development of the tubercles in fresh parts of the lungs.

(1) There may be a localized catarrhal bronchitis.

(2) There may be an inflammation of the walls of the bronchi with partial destruction of these walls, and the formation of cylindrical or sacculated bronchiectasiæ. The walls of the cavities thus formed may be converted into connective tissue, or they may remain suppurating and necrotic.

(3) There may be an interstitial pneumonia with the production of new connective tissue, the obliteration of the air spaces, and the consolidation of portions of the lung.

(4) There may be dilatation of the air spaces of the portions of the lungs which are not consolidated.

(5) There may be thickening of the pulmonary and costal pleuræ with connective-tissue adhesions.

(6) While the morbid process begins as a localized tubercular inflammation of the lung, and often retains throughout this local character, yet it may also happen that from this local lesion other parts of the body may be infected. Tubercular laryngitis and tubercular inflammation of the solitary and agminated glands of the small intestine often complicate the pulmonary lesion, and sometimes even acute general tuberculosis is produced.

Symptoms.—The disease may follow an acute pulmonary tuberculosis, or it may be chronic from the outset. There is such a very great difference in the behavior of the disease in different persons that it is necessary to arrange the cases into a number of groups according to the character of the lesions and the symptoms.

1. There seems good reason to believe that a small tubercular inflammation of one apex often runs its course and terminates in recovery, without ever giving symptoms of sufficient severity to attract the attention of the patient or lead him to consult a physician.

2. There are patients who suffer for some time from pulmonary emphysema with its attendant symptoms. In them miliary tubercles are formed in the lungs and slowly increase in number, but are not accompanied with the growth of much connective tissue, and are scattered at some distance from each other through the lungs. The formation of the tubercles does not change the physical signs belonging to the emphysema which the patient already has. The ordinary rational symptoms of emphysema continue unchanged, but the patients lose flesh and strength rapidly, and finally die very much emaciated. It is very difficult to distinguish these cases from the bad cases of emphysema without tuberculosis.

3. A very common form of the disease is that in which the inflammation is confined to one or both apices, continues for some time, subsides, and the patient recovers.

In these cases the first symptoms may be one or more small or large hæmoptyses. Before the bleeding the patients have had no pulmonary symptoms, but after the bleeding these symptoms are gradually developed.

Or the first symptom may be a troublesome cough with little or no expectoration. This cough at first does not seem of much consequence, but it continues in spite of all remedies.

Or the patients simply lose flesh and strength without any evident reason for this loss of nutrition. The pulse is also increased in frequency, but for a time there are no pulmonary symptoms.

At this early stage of the disease there may be no physical signs.

As the disease goes on the patients have more or less cough, which troubles them only in the morning, or occurs in attacks, or is persistent and troublesome through the whole day. There may be no expectoration. When present the sputa are mucous or muco-purulent, not abundant, and often contain tubercle bacilli. There is some dyspnœa on exertion. Some of the patients complain of a great deal of pain over the inflamed lung, others have no pain at all. Hæmoptyses may be repeated at any time, and are apt to be followed by an increase in the severity of the symptoms. The frequency of the pulse is increased in nearly all the patients. A febrile movement is not a prominent symptom; there may be afternoon temperatures of 100° F., followed by sweating, but often during much of the time there is no fever at all. The appetite is poor, nausea and vomiting are often troublesome. The patients lose flesh and strength. In some cases the symptoms are not at any time severe; in others the constitutional disturbances are so out of proportion to the lesion as to indicate systemic infection.

The physical signs become more marked with the further development of the thickening of the pleura, the formation of miliary tubercles, the growth of interstitial connective tissue, the localized bronchitis, and the dilatation of the bronchi. So we find: retraction of the chest wall above and below the clavicle, the percussion sound of higher pitch and of shorter duration above and below the clavicle, subcrepitant râles, friction sounds, a higher pitched and louder voice, the breathing diminished, or of altered quality, or with prolonged expiration.

Such a circumscribed tubercular inflammation usually continues for a year, sometimes longer, then it subsides and the patients recover. The portion of lung which has been inflamed is left permanently changed into connective tissue.

As the patient has had one attack of tubercular inflammation, so, although he has entirely recovered from this attack, he may have subsequent attacks of the same kind. In some of the patients, after the subsidence of the inflammation, tubercle bacilli are left in the portion of lung which has been inflamed. These may at any time later serve as a source of infection for a new local or general tuberculosis.

4. The tubercular inflammation, beginning at the apex of one lung, gradually extends and involves a large part of both lungs. The pleuritic adhesions become more extensive, a larger number of bronchi are involved in the catarrhal bronchitis, miliary tubercles and new connective tissue replace more and more of the lung tissue. The changes in the walls of the bronchi and of the lung surrounding them result in the formation of bronchiectatic cavities, which constantly increase in size, and the walls of which are necrotic or suppurating. As the disease progresses, therefore, the patients suffer, not only from the infection due to the tubercular inflammation, but also from that due to the necrosis and suppuration of the walls of cavities, while in addition more and more of the lungs is rendered unfit for breathing.

The physical signs of consolidation and of pleuritic adhesions become more marked, and as the cavities are formed and increase in size their physical signs are added.

The cough, which depends at first upon the bronchitis or the pleurisy, is made worse by the formation of bronchiectasiæ. The expectoration becomes more profuse, more purulent, and contains portions of dead lung. Bacilli are present in the sputum in large numbers. The difficulty in breathing becomes more troublesome. The patients differ very much as to the presence or absence of pain in the chest. Either large or small hæmoptyses may be repeated at any time. At first the blood comes from the mucous membrane of the bronchi, but after cavities have been formed there may be bleeding from eroded vessels in their walls. This bleeding is apt to be profuse, continuous, and often fatal. The pulse continues to be rapid through the greater part of the disease. The fever becomes higher and more continuous as the disease progresses, especially after the formation of cavities. The appetite is poor, and nausea and vomiting are often troublesome. In women, menstruation becomes irregular or ceases altogether. The patients get worse from year to year, but often with periods of improvement, and the whole duration of the disease is apt to be very considerable.

After a time, in many of the patients, a tubercular inflammation of the larynx or of the agminated glands of the small intestines is added, and then the loss of flesh and strength is much more rapid.

5. There are cases in which the tubercular inflammation gradually extends until the whole of both lungs is thickly studded with miliary tubercles and in addition there are extensive pleuritic adhesions, but there is little diffuse fibrous tissue, little or no bronchitis, and no dilatation of the bronchi.

The clinical history of these patients is very misleading. They have no cough, no hæmoptysis, no pulmonary symptoms, no fever. There are no physical signs except those belonging to the pleuritic adhesions. But very often the functions of the stomach and intestine are much disordered. The most striking symptom of these patients is their emaciation. This goes on steadily until the patients are mere skeletons, looking as if they were starving to death with a cancer of the stomach. And yet they may really be taking and retaining a considerable quantity of food. But in spite of the food they continue to lose flesh as if they had a malignant disease. The diagnosis of these cases is often extremely difficult.

6. There are cases in which a tubercular laryngitis is responsible for most of the symptoms, the lesions in the lungs being inconsiderable. The upper part of the larynx and the epiglottis are the portions usually involved. There is first a formation of tubercle granula here and there in the stroma of the mucous membrane, with more or less catarrhal inflammation. After a time the tubercle granula and the mucous membrane over them become necrotic, soften, slough, and form ulcers. These ulcers do not heal, but rather increase in size, their floor and walls being

formed partly of round-celled tissue. The mucous membrane left between the ulcers is thickened and the seat of chronic catarrhal inflammation.

With such a tubercular laryngitis the patients have a cough with muco-purulent expectoration, a changed voice, and sometimes laryngeal dyspnœa. But the thing which troubles them the most is the pain in the throat, which is made worse by swallowing. This may interfere seriously with the feeding of the patient.

The patients lose flesh and strength, but are not confined to bed. Indeed, many years may pass before the pulmonary tuberculosis gives much trouble.

7. In a few cases a very small miliary tuberculosis of the lung is complicated with an extensive tubercular inflammation of the small intestine.

These patients have a little cough, and perhaps the physical signs of a small consolidation at one apex. It is important to remember that they do not necessarily have diarrhœa. But they lose flesh and strength with a rapidity which the pulmonary conditions do not account for.

Prognosis.—In the early periods of the disease, with little evidence of systemic infection, we may often expect the complete recovery of the patient. As more of the lungs is involved, as cavities are formed, as the complicating laryngitis and enteritis are developed and the evidences of systemic infection become manifest, the prognosis is worse and worse.

Treatment.—The curative treatment of chronic miliary tuberculosis is embraced in two principal things—climate and feeding.

The selection of a proper climate is to be made with reference to the individual rather than to the condition of the lungs. It should be a climate where he feels well, eats well, sleeps well, and gains flesh and strength. If no one climate answers this purpose, the patient should travel from place to place. The climatic treatment should be continued, if possible, for two full years, and for some persons it is necessary that they should pass the rest of their lives in a favorable climate.

The feeding consists in enabling the patient to eat and digest considerable quantities of wholesome food and of fats. To do this the most minute attention is necessary to the functions of the stomach, the liver, and the intestines. Great care should be taken to avoid the use of all medicines which interfere with the patient's ability to eat and digest food. Wines and spirits will with some persons increase the appetite and the nutrition, with other persons interfere with digestion and do harm. Often we are much helped by the use of the stomach tube. Not only can we in this way cure a complicating chronic gastritis, but we can introduce into the stomach much larger quantities of fluid food than the patients are willing to swallow.

It may also be necessary to alleviate symptoms. The cough is not only annoying, but it often interferes with eating and sleeping. It is, therefore, important in each case to determine the principal cause of the cough.

It may be due to a catarrhal inflammation of the nose and throat, or to either a catarrhal or tubercular inflammation of the larynx. These conditions are best treated by local applications made with the spray or with the brush.

It may be due to the pleuritic adhesions. If this is the case, counter-irritation by blisters or iodine may be of service, but some of these pleuritic coughs can only be controlled by opium.

For the cough which is due to the bronchitis and to the cavities a great many remedies have been employed. In selecting from these remedies it is well to prefer those which do not disorder the stomach. Creosote seems to be capable of exerting a real effect on the bronchitis, but, unfortunately, it is apt to disorder the stomach. I prefer to use it by inhalation or by enema. For inhalation a mixture is made of equal parts of creosote, chloroform, and alcohol. The sponge of a naso-oral inhaler is moistened with a few drops of this mixture, and the patient wears the inhaler all the time except when sleeping or eating. An enema is made of five to twenty drops of creosote with some white of egg and a little water, and this can be used once a day. The different preparations of tar and of turpentine, terebene and terpine hydrate, seem to be of service in some cases. The methodical inhalation of compressed air is highly thought of by some physicians. All sorts of combinations of opium, ipecac, squills, sanguinaria, hydrocyanic acid, chloroform, senega, etc., are given as cough mixtures. The mineral acids, nux vomica, and potassium iodide may somewhat control the bronchitis.

If the patients are anæmic they may be benefited by one of the preparations of iron.

The hemorrhages from the bronchi may be small or large, but even if large and continued for several days they are very seldom fatal. They do, however, weaken the patient very much, and are often followed by an extension of the tubercular pneumonia. To check such hemorrhages it is customary to use hypodermic injections of morphine, of adrenalin chloride, or of ergotin, or to give by the mouth five grains of gallic acid every two hours, twenty drops of fluid extract of hydrastis every three hours, or one grain of ipecac every hour.

The hemorrhages which come from eroded vessels in the walls of cavities cannot be controlled.

The fever and night sweats may be made less severe by the use of antifebrin or phenacetin, alone or combined with arsenic or quinine; by the mineral acids, belladonna, or the oxide of zinc; by sponging off the body with hot water at night.

For the diarrhœa we employ a restricted diet and a number of drugs. The drugs most frequently employed are: the preparations of mercury, ipecac, iron, arsenic, camphor, acetate of lead, bismuth, castor-oil, opium, and naphthalin. These drugs are used singly or combined in different ways.

ACUTE PULMONARY PHTHISIS.

The clinical characters of acute phthisis are sufficiently well known. A person who has previously been in good health, or who has already suffered from chronic phthisis, either suddenly or after a few days of indisposition, becomes seriously ill. There is a well-marked febrile movement and a rapid pulse, cough, expectoration, the physical signs of consolidation of the lung, and sometimes hæmoptysis. Rapid loss of flesh and of strength follows. Most of the patients die at the end of a few weeks or months; a few recover from the immediate attack and go on to certain. These different products are found in variable relative proportion in different lungs and in different parts of the same lung. Some vesicles will be entirely filled with the granules, others with fibrin, others with epithelial cells; while others will contain granules and epithelium, or epithelium and fibrin, with an admixture of pus cells.

This diffuse hepatization seems to have no tendency to cheesy degeneration or necrosis. The products of inflammation within the vesicles may undergo fatty degeneration, but the walls of the vesicles remain intact and the blood vessels pervious. If we can judge from clinical experience and physical signs, this diffuse hepatization is capable of resolution and may entirely disappear. If, however, the patient goes on to have chronic phthisis, then the diffuse hepatization undergoes marked changes.

II. *The White or Yellow Nodules.*—These nodules vary in their size, their shape, and their structure. They may be close together or widely separated. Some are surrounded by diffuse hepatization, others by nearly normal lung. Even those which present the same appearance to the naked eye are not all of the same nature.

They seem to be formed in air vesicles, in single air passages, in groups of air passages, and in and around small bronchi.

1. *The smallest nodules.* These are not larger than a single air vesicle or part of an air passage. In the fresh lung it is not easy to distinguish them from the diffuse hepatization by which they are surrounded. When the blood vessels have been artificially injected, the nodules are more readily recognized. These little nodules consist of a single air vesicle or part of an air passage filled with tubercle tissue, or filled partly with tubercle tissue, partly with epithelium, fibrin, and pus. The walls of the air vesicle or air passage remain intact and its vessels permeable. Such little nodules may be numerous and close together. When surrounded by air vesicles filled with products of inflammation, they constitute a peculiar hepatization, partly tubercular, partly inflammatory.

2. Nodules a little larger than these, but of the same character, are composed of several air vesicles, or of part of an air passage and air vesicles. The cavities of the vesicles are filled in the same

way with tubercle tissue, but in addition the walls of the vesicles are affected. They become split up and changed into tubercle tissue. As this process goes on, the outlines of the vesicles are lost and the blood vessels in their walls can no longer be injected. Such nodules are identical in their structure with the miliary tubercles seen in acute miliary tuberculosis. It sometimes happens that a section will show the whole of an air passage filled with tubercle tissue in this way. The centres of these miliary tubercles may undergo cheesy degeneration in the ordinary way.

3. *The peribronchitic nodules.* These nodules surround either partly or completely a bronchus or an air passage. Their size and shape vary with the size of the bronchus, the direction in which it is cut, and the number of surrounding air vesicles involved.

When we examine these peribronchitic nodules more closely, it becomes evident that they do not all have the same structure.

(*a*) Some of the nodules consist simply of a bronchus filled with pus and epithelium, its walls infiltrated with cells, and of a number of air vesicles filled with epithelium, fibrin, and pus. Such nodules are identical in their structure with the peribronchitic nodules which are formed in the ordinary broncho-pneumonia of children and adults.

(*b*) Other nodules are composed of a bronchus filled with pus and epithelium. The wall of the bronchus is infiltrated with small cells or with tubercle tissue. Around the bronchus is a zone of air vesicles filled with tubercle tissue, the walls of the vesicles split up and only visible with high magnifying powers. Around this tubercular zone is another outer zone of vesicles filled with epithelium, fibrin, and pus.

(*c*) Other nodules are composed as before of a bronchus with infiltrated walls, and filled with inflammatory products. Around the bronchus is a continuous zone of tubercle tissue, in which the walls of the vesicles cannot be seen. Artificial injections of the blood vessels penetrate only very imperfectly into this tubercular zone. Outside of this tubercular zone will be a zone of vesicles filled with epithelium, fibrin, and pus, or with tubercle tissue. Very often the contents of the bronchus, the walls of the bronhave the history of chronic phthisis; a few recover and live f many years.

After death we find a considerable portion of one or both lun consolidated, but this consolidation is not of a uniform appe ance. The larger part of the consolidation looks like ordina red hepatization, or else it is of a peculiar grayish color, differe from the gray hepatization of lobar pneumonia. In this diffu hepatization are other portions of solidified lung of differe appearance. They are of a white or yellow color, much dens than the surrounding hepatization, or they may be softened their centres. These white solidifications are of many differe sizes and shapes. The smallest are hardly larger than an vesicle, the largest may include a considerable part of a lol Their size and shape are made more evident if the blood vess are artificially injected, for into these nodules the injection do not run.

Many of the smallest of these whitish masses are scatter irregularly through the lung tissue like miliary tubercles. will be seen later, they really are miliary tubercles.

Other similar masses, also of small size, are situated regularl around small bronchi. When this is the case, the cavities of th bronchi may be filled with inflammatory products and the walls infiltrated.

Still other masses will correspond to a single air passage, or a group of air passages, looking as if they were produced some cause which affected definite and isolated portions of lung.

Other masses, again, are so large that they have the appeara of a diffuse infiltration of a considerable portion of the lu But these large infiltrations seem to be made up of a num of smaller masses.

If the patient lives long enough after the development of disease, many of these masses, both large and small, will be fou degenerated and softened.

The bronchi of the affected lung are regularly the seat morbid changes. They may be congested and coated with muc they may be filled with pus, epithelium, and fibrin, and th inflammatory products may undergo cheesy degeneration; t may be irregularly dilated, so as to form cavities of some size; or a great number of the smaller bronchi may be symmetrically dilated, so that on section the lung looks honeycombed.

The pathological condition is really a complex one, and we gain nothing by trying to make it appear more simple than it really is.

It is necessary to examine each of the different parts of the lesion in detail and see what their real structure is.

I. *The Diffuse Hepatization.*—It must be remembered that we are considering the diffuse hepatization of acute phthisis, not that of chronic phthisis, and especially the conditions found in the lungs of adults. This hepatization presents the gross appearance of the red hepatization of lobar pneumonia, except that it is smoother; or it is of a peculiar gray color, sometimes almost gelatinous-looking. It does not seem to degenerate or to soften except immediately around some of the nodules, of which we shall speak presently.

The blood vessels in the walls of the air vesicles can be easily filled with an artificial injection. The walls of the air vesicles show no marked changes in their substance, although their surfaces are overlaid with new cells.

The cavities of the air vesicles are filled with inflammatory products, but this filling is not uniform. Some vesicles are filled and even distended, while others only contain a small amount of such new substances. These inflammatory products are: pus cells, fibrillated fibrin, large, polygonal, nucleated cells resembling epithelium, minute, shining granules, and a peculiar transparent matter. The pus cells have the ordinary appearance of these bodies, varying in their size and in the character of their cell substance, some being finely, some coarsely granular, and some fatty. The fibrin has the same appearance of a network of fibrils as is seen in acute lobar pneumonia. The large, polygonal cells resemble, in shape and appearance, epithelial cells, but some of them undergo degenerative changes, becoming larger, globular, coarsely granular or fatty, or very large and transparent. The granules are small, shining particles, not dissolved by acetic acid, liquor potassæ, or chloroform, and are stained either with methyl violet or hæmatoxylin. Their nature is uncertain. These different products are found in variable relative proportion in different lungs and in different parts of the same lung. Some vesicles will be entirely filled with the granules, others with fibrin, others with epithelial cells; while others will contain granules and epithelium, or epithelium and fibrin, with an admixture of pus cells.

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(*d*) In other peribronchitic nodules, although the cavity of the bronchus contains inflammatory products, yet its wall will remain unchanged, and around the bronchus will be air vesicles filled with tubercle tissue. Such nodules seem to be peribronchitic miliary tubercles. It will be observed that in all these peribronchitic nodules a large part of the consolidation is due to changes in the air vesicles.

In connection with peribronchitis it is convenient to speak of two lesions which affect the bronchi directly in some cases of acute phthisis.

1. The walls of some of the larger bronchi are infiltrated with tubercle tissue which undergoes cheesy degeneration. The cavity of the bronchus is dilated, and contains inflammatory products also in a condition of cheesy degeneration, The adjacent air vesicles may be unchanged, or may contain tubercle tissue, or pus, fibrin, and epithelium. This change affects especially the larger and medium-sized bronchi, sometimes a large bronchus and its branches. The dilatation may be sufficient to form a cavity of some size, which may be further enlarged by the disintegration of the cheesy bronchial wall. For the most part in these bronchi the cheesy degeneration of the wall is so advanced that no structure can be made out, but sometimes one can find a place which seems to show the previous infiltration of tubercle tissue.

2. There may be a general dilatation of most of the bronchi in a considerable portion of the lung without any marked change in their walls, and with only a moderate amount of inflammatory products in their cavities. This change is especially apt to affect the medium-sized and small bronchi. The lung tissue between the dilated bronchi is usually consolidated. When such a lung is cut it looks as if it were honeycombed with small cavities, but these cavities are only sections of the dilated bronchi.

The larger nodules found in acute phthisis seem to be of three kinds.

1. Peribronchitic nodules such as have been already described.

2. Bodies resembling miliary tubercles in their structure, but much larger—the "tubercules massifs" of French writers. These

nodules have just the same structure as many of the ordinary miliary tubercles already described, but they are on a larger scale, and the centre is regularly the seat of cheesy degeneration.

3. Nodules formed by a number of air vesicles in the condition of coagulation necrosis.

The areas of coagulation necrosis are small or large, scattered at considerable intervals or close together. In each one there is a centre of air vesicles filled with inflammatory products in the condition of coagulation necrosis, and surrounded either by a zone of ordinary red hepatization or by a zone of tubercle tissue.

If we look more closely at these areas of coagulation necrosis, we see that their vascular supply is entirely cut off and that artificial injections do not penetrate them. The outlines of the walls of the vesicles are visible, but the walls are thin, transparent, recognizable principally by their elastic fibres. The cavities are filled with polygonal cells and shrivelled nuclei. The cells are pale, shining, devoid of nuclei, and have the characteristic appearance of cells in the state of coagulation necrosis. In some vesicles, however, there is nothing but coagulated fibrin. These vesicles filled with fibrin are not as opaque as the others, so that a mottled effect is produced.

There is nothing especial to be noticed in the zone of ordinary hepatization which surrounds the necrotic centres. The cavities of the vesicles are filled with epithelium, pus, fibrin, and granular matter in varying proportions. The walls of the vesicles are unchanged, their vessels are pervious. It is to be noticed that there is no appearance of a gradual transition between the necrotic centres and the hepatized peripheries. There are no vesicles giving pictures of coagulation necrosis in different stages. All the necrotic portion is in the same condition, and is separated abruptly from the surrounding hepatization.

In those nodules which have a peripheric zone of tubercle tissue, the necrotic centre is sharply separated from the tubercular zone, and this tubercular zone gives an indistinct appearance of being made up of altered air vesicles.

If we examine this tubercular zone more closely, we find that there are changes in the walls of the air vesicles and in their cavities. The walls are changed into tubercle tissue. At first the blood vessels in the walls remain pervious, but as the transformation of the walls becomes more complete the vessels disappear. The cavities of the vesicles are partly or completely filled with tubercle tissue, epithelium, and pus. The tubercle tissue grows inward from the walls of the vesicles, or is formed in their cavities; it has the regular structure of a delicate basement substance, more or less reticulated, provided with nuclei, and enclosing spaces containing large, polygonal, nucleated cells. Giant cells are sometimes present, but are not constant. The process seems to be of the same character as the formation of diffuse tubercle tissue already described in connection with chronic miliary tuberculosis.

These nodules may remain in the condition just described up to the time of the patient's death. But in some cases they undergo further changes. Cheesy degeneration begins at the centre of the necrotic portion of the nodule, involves the whole of this portion, and may extend to the surrounding zone of hepatization or of tubercle tissue. The nodules will then present quite a different appearance. There is a centre composed of an amorphous cheesy mass, in which no structure can be made out, and around this is a zone of tubercle tissue or of hepatization. In this condition it is often difficult to distinguish these nodules from the peribronchitic nodules and the "tubercules massifs."

In such a condition of dry cheesy degeneration a certain number of the nodules may remain indefinitely; but in others the degenerated portions will soften, and in this way cavities will be formed of sizes corresponding to the original nodules; or, if the nodules are close together, several small cavities will join to form larger ones. In this way we sometimes find a large part of a lung honeycombed with small cavities having ragged walls; the number of cavities corresponding with the number of the areas of coagulation necrosis.

Judging from the results of Prudden's experiments on the injection of tubercle bacilli into the air tubes of animals, it seems probable that these areas of coagulation necrosis are due to the inhalation of considerable numbers of tubercle bacilli into groups of air spaces, where they act as direct irritants, the disposition to necrosis being rendered still greater by an additional infection with streptococci.

It seems evident, then, that in acute phthisis we have a lesion of peculiar character. It is very far from being a simple broncho-pneumonia with degenerative changes, or an acute miliary tuberculosis combined with pneumonia. The lesions are much more complex. Both the bronchi and the parenchyma of the lung take a share in the morbid process. The lesion seems to be an inflammatory one, the products of inflammation being pus, epithelium, fibrin, and tubercle tissue; but it is complicated by the formation of areas of coagulation necrosis. The tubercle tissue is arranged partly so as to form miliary tubercles and large tubercles, partly as peribronchitic nodules, partly as tubercular zones around areas of coagulation necrosis. The prognosis seems to depend mainly upon the number and size of the areas of coagulation necrosis, and it seems probable that most of these are formed at the very outset of the disease. The diffuse pneumonia may undergo resolution, the miliary tubercles and peribronchitic nodules do not occupy much of the lung and may be transformed into cheesy material or fibrous tissue; but the areas of coagulation necrosis destroy a considerable part of the parenchyma of the lung and have a natural tendency to soften and form cavities.

Symptoms.—The invasion may be acute or subacute.

1. The acute cases. The patients are suddenly attacked with chills, fever, pain in the side, cough with mucous expectoration, and marked prostration. The appearance of the patient is like that of a person attacked with lobar or broncho pneumonia, and we are often in doubt at first as to the true nature of the disease. One symptom of the invasion, however, is not often seen except with phthisis, and that is the bleeding from the bronchi. For a day, or for several days, many of the patients cough up very considerable quantities of blood. Within a few days we begin to get the physical signs, which will be found to vary according to the anatomical condition of the lung. If there is complete consolidation of one or more lobes with fibrin on the pleura, we get dulness on percussion, bronchial voice and breathing, and subcrepitant and crepitant râles. If there is only broncho-pneu-

monia without consolidation, we get sibilant and sonorous breathing and coarse and subcrepitant râles. If there is broncho-pneumonia with areas of diffuse consolidation, we get sibilant and sonorous breathing and coarse and subcrepitant râles, and small areas over which dulness on percussion, increased voice, and a crepitant râle are found. The patients continue seriously ill and with high temperatures for one or two weeks. Then there is a partial subsidence of the symptoms. After this the patients may:

(*a*) Continue to get worse. The fever continues, the cough is very troublesome and accompanied with muco-purulent expectoration, there is rapid loss of flesh and strength, the patients pass into the typhoid state with alternating delirium and stupor. The physical signs which existed earlier in the disease continue, and there are added the coarse and gurgling râles which accompany the dilatation of the bronchi and the softening of the dead areas of consolidation. These patients die at the end of a few weeks or months.

(*b*) Improve very considerably. The temperature falls, the expectoration diminishes, the cough is less frequent, the appetite returns. The patients gain flesh and strength, they are able to leave the bed; and later the house, but yet they are not well. A considerable portion of the lung remains diseased and the patients go on to the condition of chronic phthisis.

(*c*) Recover. Of the lesions of acute phthisis, the tubercular changes, the death of portions of lung, and the dilatation of the bronchi are necessarily permanent. On the other hand, the catarrhal bronchitis may subside, the epithelium, pus, and fibrin within the air spaces may degenerate and be absorbed. It is possible, therefore, for the patients who have only catarrhal bronchitis, exudative pneumonia, and comparatively little tubercular pneumonia to recover. We see this in two sets of cases. First, the patients who have consolidation of an entire lobe, due to the filling of the air spaces with epithelium, pus, and fibrin, and to the presence of miliary tubercles. The epithelium, pus, and fibrin can be absorbed, the tubercles are converted into fibrous tissue, and the patients get well with a lung which is normal except for the presence of fibrous nodules. Second, the patients who have tubercular broncho-pneumonia without destruction or dilatation of the bronchi, and without areas of diffuse consolidation. These patients can get well with lungs which are normal except for the presence of a number of peribronchitic fibrous nodules.

2. The subacute cases. The extent of lung at first involved is small, but gradually increases in size. The patients usually have cough with mucous or muco-purulent sputa which contain bacilli, but sometimes there is very little either of cough or of expectoration, and that even while cavities are being formed. The difficulty in breathing increases with the extent of lung involved, pain over the chest is present in some cases, absent in others.

There is regularly a rise of temperature in the afternoon with sweating at night, and the temperature is higher and more continous after cavities, with suppurating walls, are formed. But we have to become accustomed to very great discrepancies between the lung lesion and the height of the temperature, and even to find no fever with an advancing consolidation of the lung.

There may be bleeding from the bronchi or from eroded vessels in the walls of cavities. A large hemorrhage from the bronchi is apt to precede the inflammation of a fresh portion of the lung. The patients, as a rule, have no appetite and gradually lose flesh and strength. But in some persons the changes in the lung for some time produce very little effect on the general health. And it is sometimes very curious, and of importance in judging of treatment, to see patients eating well and gaining flesh with consolidation of an entire lung and without any real improvement in their tubercular pneumonia.

The physical signs are those of the pleurisy, the consolidation, the bronchitis, and the cavities.

The patients are for the most part not sick in bed and the disease progresses either continuously or in attacks.

Some of the patients go on to have chronic phthisis. Some of them recover, [illegible] lungs more or less permanently damaged. [illegible] permanently [illegible]

[illegible] and Lobar Pneumonia.—Female [illegible]

[illegible]

began to cough. After a few days there was muco-purulent sputum. For seven days there has been fever every evening.

Now, on the twelfth day, there are dulness and subcrepitant râles over the whole of the right lung. She continued in the same condition, with afternoon fever and steady loss of flesh and strength, until she died on the thirtieth day.

Autopsy.—The brain was not examined.

The heart, liver, kidneys, and intestines are normal.

The right lung is entirely consolidated with red and gray hepatization mottled with small miliary tubercles. In the left upper lobe there are the same changes, but to a less extent.

In the diffuse hepatization the air spaces are filled with epithelial cells, pus cells, and granular matter.

CASE VII. *Acute Broncho-Pneumonic Phthisis; Recovery; Death after four years from Cancer of the Common Bile Duct.*—Female, 39 years old, January 14, 1884.

Seventeen years ago she had a single hæmoptysis, not followed by any illness. Eight years ago she had another hæmoptysis. Four years ago another hæmoptysis, followed by pain in the lower lobe of the left lung.

In January, 1880, she had another hæmoptysis, followed by cough and expectoration, afternoon fever, pain in the left chest, with dulness and bronchial breathing over the lower lobe of the left lung.

She continued in the same condition until March, when she began to improve, and by June there were no more constitutional symptoms or physical signs.

She continued well and without pulmonary symptoms until July, 1883, when she began to lose flesh and strength.

In October, 1883, jaundice appeared and continued.

In January, 1884, she came back to the hospital with bad obstructive jaundice. There were no physical signs over the lungs. March 15, 1884, she died.

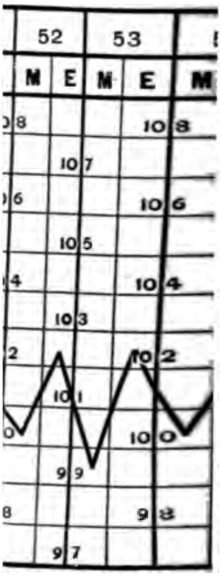
Autopsy.—The body is emaciated and jaundiced.

The brain was not examined.

The heart is small.

The lower lobe of the left lung is small. The pulmonary pleura is thickened and the lung puckered. Scattered through this lobe are little, cheesy nodules which look like small bronchi, filled with cheesy pus and surrounded by vesicles with obliterated blood vessels and cheesy contents. Around these nodules are zones of connective tissue well supplied with blood vessels.

There is a small carcinoma of the common bile duct with secondary nodules in the liver.



HTHISIS.

CASE VIII. *Acute Broncho-Pneumonic Phthisis with Miliary Tubercles and Diffuse Consolidation.*—Male, 33 years old, August 19, 1894.

He says that he was well until two weeks ago; then he woke up in the night with a stitch in the left side. The pain continued, with prostration, fever, dyspnœa, cough and scanty sputum. Now, on the fifteenth day, he is fairly nourished. Over the whole left lung there are dulness on percussion, subcrepitant râles, and bronchial breathing.

The temperature ran from 98° to 100° F. in the morning and from 102° to 105° F. in the evening, the pulse from 96 to 134, the breathing from 26 to 44.

On the fiftieth day he developed thrombosis of the left femoral vein.

He got steadily worse and died on the seventy-fifth day.

Autopsy.—The body is emaciated.

The brain was not examined.

The heart is normal.

The left lung is completely consolidated—diffuse red hepatization mottled with small white areas. In the diffuse hepatization the air spaces are filled with epithelial cells. The white areas are, some of them, composed of small bronchi with thickened walls and zones of peribronchitic pneumonia, while others are miliary tubercles.

The right lung is congested and œdematous; it is studded with miliary tubercles and small areas of broncho-pneumonia.

The liver is fatty.

The spleen is large and soft.

The kidneys are normal.

The stomach shows the changes of chronic gastritis.

The intestines are normal.

There is a large red thrombus in the left external iliac and femoral veins.

CASE IX. *Acute Phthisis with Necrosis.*—Male, 27 years old, October 24, 1882.

Four weeks ago he began to have chills, followed by fever every second night. Three weeks ago he began to cough with scanty sputum.

Now, at the end of four weeks, his tongue is dry and coated, bowels regular; urine 1025, no albumin. Over the whole left lung behind there are dulness on percussion, bronchial voice and breathing, and subcrepitant râles. The patient continued to lie quietly in bed, with flushed face, taking very little notice of anything, until he died at the end of four months.

(For Chart of Case IX, see Fig. 71.)

Autopsy.—The brain was not examined.

The heart is normal.

The stomach, spleen, and kidneys are normal.

In the ileum the lowest Peyer's patch is swollen and contains miliary tubercles.

The left pleural cavity is one-third full of turbid serum; the pulmonary pleura is coated with fibrin. The whole left lung is completely consolidated—diffuse hepatization, cheesy nodules, and small cavities with ragged walls. The upper lobe of the right lung is in the same condition.

CASE X. *Acute Phthisis; Rapid Formation of Bronchiectasia.*—Female, 24 years old, October 16, 1883.

Seven weeks ago she began to cough with profuse sputum, followed by night sweats and rapid loss of flesh and strength.

Two weeks ago she began to have diarrhœa, which continued until two days ago.

Now she is feeble and emaciated, with cough and profuse muco-purulent sputum. There are dulness on percussion and coarse and subcrepitant râles over the whole left lung.

The temperature ran irregularly between 98° and 103° F., she had diarrhœa, became stupid, and died after being sick eleven weeks in all.

Autopsy.—The brain was not examined.

The heart is normal.

In the right lung there are some old, hard miliary tubercles near the centre of the lung.

The left lung is completely consolidated with diffuse hepatization and large and small cheesy nodules. In the upper lobe are two large cavities communicating with bronchi. The lower lobe is honeycombed with cavities corresponding to large bronchi with cheesy walls.

The liver, spleen, and kidneys are normal.

The lower part of the ileum and the upper part of the colon are congested and coated with fibrin.

The *Prognosis* of acute phthisis is unfavorable. Complete recoveries are rare, but the number of patients who go on living with damaged lungs for many years is considerable.

Treatment.—While the inflammation of the lungs is active the patients are to be kept in bed, on a fluid diet, the hæmoptysis controlled by ergot, hydrastis, or ipecac, the patients made more comfortable by opium or the bromides. The preparations of creosote are of service.

As the acuteness of the inflammation subsides, the patients return to solid food, they get out of bed, and the question of a suitable climate for them has to be determined.

In some cases it is evident that the changes in the lungs are so extensive and profound that no improvement can be expected; these patients are best kept at home.

In some cases the extent of lung involved is comparatively small, so that we may hope for complete or incomplete recovery. The proper climate for these patients is a dry, inland one where they are not likely to have fresh attacks of inflammation of the lung. Whether this climate should be a warm or a cold one must be determined by the character of the individual. In the cases of tubercular broncho-pneumonia without consolidation a cold, dry, inland climate seems to be the best.

CHRONIC TUBERCULAR PHTHISIS.

Definition.—A chronic affection of the lungs characterized by tubercular inflammation associated with productive and exudative inflammation.

Etiology.—Chronic phthisis regularly succeeds acute or subacute phthisis, but it may also follow acute or chronic miliary tuberculosis of the lungs, or a tubercular inflammation of some other part of the body.

Morbid Anatomy.—The changes in the lungs are of the same nature as those found in acute phthisis, but modified by the long duration of the inflammation. The pleura is coated with successive layers of fibrin, or thickened by the growth of new connective tissue, or covered with adhesions. More or less of the lung is consolidated. This consolidation is effected partly by the filling of the cavities of the air spaces with inflammatory products, partly by a growth of new tissue in the walls of the air spaces and between them.

When the hepatization is effected only by changes within the cavities of the air spaces, the affected portions of lung are solid, increased in size, of red, gray, white, or yellow color. When the hepatization is due to interstitial inflammation the affected portion of lung is dense, but may still be partly aërated; it is diminished in size and looks like fibrous tissue or granulation tissue; it is often changed in color by the deposition of black pigment. The combination of intra-alveolar and interstitial pneumonia with dilatation of the bronchi and the formation of cavities gives a great variety of pictures.

We find some of the air spaces filled with large epithelial cells either well formed or fatty; some with an amorphous granular matter or a peculiar translucent coagulated substance; some with fibrin, pus, and epithelium either fresh or in the condition of cheesy degeneration; some with new connective tissue. The walls of these air spaces remain unchanged, or they are compressed and the blood vessels obliterated, or they are thickened.

The interstitial inflammation affects the walls of the air spaces, the bronchi, the blood vessels, and the septa between the lobules. It results in the production of new connective tissue, of round-celled tissue, and of tubercle tissue, either separately or together. By this growth the air spaces are compressed, deformed, and obliterated in a variety of ways.

The walls of some of the bronchi are infiltrated with round cells or with tubercle tissue. This infiltration is not symmetrical, but affects a bronchus in some particular portion of its length, and in this portion some parts of the circumference of the bronchus are affected more than others. As a result of this irregular infiltration the wall of the bronchus yields here and there, and small sacculated dilatations are formed. In some cases, especially in chronic miliary tuberculosis, the cavities thus formed simply become larger and larger, compressing the surrounding lung. More frequently, however, the process extends from the wall of the bronchus to the surrounding air vesicles, so that the bronchus is surrounded with tubercle tissue, round-celled tissue, and air vesicles filled with inflammatory products. Then necrosis sets in, with softening of the walls of the bronchus and of the surrounding inflamed lung. So cavities are formed partly by destruction of tissue, partly by dilatation of bronchi of which the walls are either necrotic or suppurating.

Some of the cavities in chronic phthisis seem to be formed simply by the softening of areas of coagulation necrosis, but the larger number are bronchiectasiæ such as have just been described.

The cavities which are formed by dilatation without necrosis of their walls may be developed with hardly any cough or expectoration. As we examine the patients from time to time, the change in the percussion and the breathing show the increasing size of a cavity which remains nearly dry and empty.

The cavities formed both by dilatation and necrosis, on the contrary, are regularly accompanied by an harassing cough and profuse expectoration. They contain pus, mucus, fragments of dead tissue, and great numbers of tubercle bacilli. Their walls are ragged and irregular, partly necrotic, partly suppurating. In this active condition the cavities may remain up to the time of the patient's death. Or, instead of this, the active processes may subside, the production of pus and the death of tissue cease, the cavity become dry and its walls changed into fibrous tissue. The natural tendency of all these cavities is to increase in size and open into each other.

Miliary tubercles are scattered through the inflamed lung in varying numbers.

The tubercle bacilli are found principally in the walls of the cavities and in the inflammatory products which have undergone cheesy degeneration.

Symptoms.—The cough depends principally upon the bronchitis and the morbid processes going on in the walls of the cavities. The expectoration consists of mucus, pus, and fragments of dead lung tissue, with many tubercle bacilli. But in patients with consolidation of the lung without bronchitis, and with cavities of which the walls are comparatively healthy, the cough and expectoration amount to very little. Generally speaking, however, the quantity of the sputa and the number of the bacilli are a fair test of the activity of the morbid process.

Hæmoptysis occurs in a large proportion of the cases, and at any time in the course of the disease. After some of these bleedings the patients feel more comfortable, but after others there is a rapid extension of the disease. Hemorrhages from eroded vessels in the walls of cavities are very dangerous.

In some of the patients the pleurisy from time to time gives pain. In others there is a considerable exudation of serum in one of the pleural cavities, which increases the difficulty in breathing. The softening of a dead portion of lung or the rupture of the wall of a cavity just beneath the pleura may cause perforation, pneumothorax, and then either a pleurisy with effusion or an empyema. At the time of the perforation the patients feel as if something had given way, and at once suffer from the most urgent dyspnœa. The heart's action becomes rapid and feeble, and the veins throughout the body congested. The characteristic physical signs are soon evident. Such a perforation usually proves fatal within a few days or weeks.

The dyspnœa on exertion increases with the extension of the disease and the consequent diminution in the extent of lung available for breathing.

Tubercular laryngitis occurs later in the disease with chronic phthisis than with chronic miliary tuberculosis. It gives rise to cough, hoarseness, and pain.

The fever and the sweating at night seem to be related to the severity of the bronchitis, and of the necrosis and suppuration of the cavities. The temperature rises and falls according to the activity of these conditions. The consolidation of the lung alone can extend with little or no fever.

The functions of the stomach and liver are sooner or later affected, either with or without chronic catarrhal gastritis and fatty infiltration of the liver. Loss of appetite, repugnance to food, nausea, vomiting, and gastric pain may seriously annoy the patient.

Toward the close of the disease a severe diarrhœa often sets in, and the patients lose flesh and strength very rapidly. In these patients after death we may find tubercular ulcers of the small intestine or only a catarrhal colitis. It may also happen that extensive tubercular ulcers of the small intestine exist without any diarrhœa at all.

Tubercular meningitis, or peritonitis, or nephritis may occur as complicating inflammations.

Chronic degeneration of the kidney, or chronic nephritis with or without exudation, are often developed after the phthisis has lasted for some time.

In women menstruation is either irregular or stops altogether.

The physical signs with only a small area of lung consolidated are: dulness on percussion, broncho-vesicular or feeble breathing, increased vocal resonance, increased bronchial whisper, and, in addition, subcrepitant râles and friction sounds. As more of the lung is consolidated the dulness on percussion becomes more marked, the voice and breathing approach more nearly to the bronchial character, and an increasing bronchitis gives coarse and subcrepitant râles. After cavities have been formed the percussion sound changes to flatness, tympanitic resonance, or the cracked-pot sound. The breathing and voice remain of bronchial quality or become cavernous. There may be gurgling râles.

The patients regularly lose flesh and strength in proportion to the extent of lung diseased. But it is not rare to see persons, who are well nourished and comparatively strong, with extensive changes in the lungs.

If we compare chronic miliary tuberculosis with chronic phthisis, we may say that in the former there are but few tubercle bacilli in the sputa or in the lungs, but little necrosis or suppuration, and less consolidation of the lung, but a loss of health and nutrition much greater than would be expected from the extent of the pulmonary lesion.

In chronic phthisis, on the other hand, the number of tubercle bacilli in the sputa and in the lungs is large, necrosis and suppuration are regularly present, the patients seem to suffer rather from septic than from tubercular infection, the severity of the symptoms is usually in direct relation with the extent of lung inflamed.

Prognosis.—It is possible for the inflammatory and necrotic processes which belong to chronic phthisis at any time to cease. When this happens, all symptoms of pulmonary disease may also cease and the patients are apparently cured. The portions of lung, however, which have been destroyed or converted into fibrous tissue are never replaced by lung tissue, so that the injury inflicted on the lungs is a permanent one. The fibrous tissue and cheesy masses left behind after the subsidence of active changes are liable to act as foci, from which fresh attacks of inflammation and fresh infection may proceed. The prognosis is, therefore, unfavorable, although life may be prolonged in comparative comfort for many years.

Treatment.—Cases of chronic phthisis are to be managed in the same way as are the cases of chronic miliary tuberculosis.

I think that such patients do best in a dry, inland climate where the altitude is not very high, and they should reside permanently in such a climate.

The patients with a good deal of bronchitis, or with suppurating cavities, may be much benefited by the continuous inhalation of creosote.

One should be particularly careful in advanced cases of this kind not to send the patients away from home to die.

SYPHILITIC PNEUMONIA.

In children who have inherited syphilis, a number of inflammations of different parts of the body are liable to be developed soon after birth. The lungs are not exempt. They may be studded with gummy tumors of different sizes, or they may be the seat of interstitial or of intra-alveolar pneumonia.

The interstitial pneumonia may cause the consolidation of one or more lobes. The section of such a consolidated lobe is smooth and of grayish or white color. The consolidation is effected by a growth of new tissue in the walls of the air spaces, the bronchi, and the blood vessels, together with the filling of some of the air spaces with epithelium.

The intra-alveolar pneumonia also produces a whitish hepatization of considerable portions of the lung. But the consolidation is effected entirely by the filling of the air spaces with fatty epithelium.

The clinical symptoms in these children are obscure. They often have other syphilitic lesions besides those in the lungs, and become weaker and more emaciated from day to day, without any pulmonary symptoms except the physical signs of the consolidation.

In adults, syphilitic inflammations of the lung are rare. The best collection of cases that I have seen is that given by Hiller in the *Charité Annalen* for 1884. He gives 58 cases of unmistakable syphilitic pneumonia.

Morbid Anatomy.—The inflammation is of productive character with the formation of round-celled tissue, of gummy tumors, and of new connective tissue. There may also be some exudation into the air spaces and a formation of epithelial cells in the air spaces. The new tissue is formed in the walls of the bronchi, the walls of the air spaces, the walls of the blood vessels, the septa between the lobules, and the pleura. The new tissue is of low vitality and may become necrotic. There results from such an inflammation, therefore: stenosis, ulceration or dilatation of the bronchi, consolidation of parts of the lung, obliteration of the blood vessels, lobulation of the lung, and thickening of the pleura.

The gross appearance of the lungs will accordingly vary in different cases.

1. There is an interstitial inflammation beginning at the root of the lung around the large bronchi and blood vessels. This causes stenosis of the bronchi, consolidation of the lung, or masses of fibrous tissue along the lines of the bronchi. The cases vary as to whether the stenosis of the bronchi or the consolidation of the lung is the principal feature.

2. The inflammation follows the type of a broncho-pneumonia with thickening of the walls of the bronchi and small zones of peribronchitic pneumonia.

3. There are large or small irregular masses, or bands, of dense fibrous tissue in any part of the lung. These replace the lung tissue, and in them may be cavities formed by the dilatation of the bronchi.

4. With the interstitial pneumonia there may be associated the formation of gummy tumors, or an obliterating endarteritis with areas of necrosis.

5. With more or less interstitial pneumonia at the roots of the lungs there is a syphilitic inflammation of the walls of the large bronchi, the trachea, and the larynx. These walls are thickened in some places, ulcerated in others, so that in some places there is stenosis, in others dilatation.

Symptoms.—Syphilitic pneumonia is one of the later manifestations of syphilis, in most of the cases coming on several years after the initial lesion. A great many of the patients have other syphilitic lesions—a fact of much assistance in making a diagnosis, which is always difficult.

Of the pulmonary symptoms perhaps the most constant is dyspnœa. This is like any dyspnœa due to narrowing of the trachea or large bronchi. First a dyspnœa on exertion, then a constant dyspnœa made worse by the least bodily or mental exertion, and becoming more and more distressing up to the time of the patient's death.

Cough is present at some time in most of the cases—a dry cough, a laryngeal cough, or a cough with mucous or muco-purulent expectoration. Small or large hæmoptyses occur from time to time in some cases.

Pain, referred to some part of the chest, is present in some cases, absent in others.

A febrile movement seems to be the exception rather than the rule.

If a syphilitic laryngitis exists the symptoms belonging to this will be added to those of the pneumonia.

The physical signs vary with the exact condition of the lungs and are often obscure. They depend upon the pleurisy, the bronchitis, the stenosis or dilatation of the bronchi, and the consolidation of the lung.

So in the different cases we may get tubular breathing over one or both lungs, absence of breathing over one lung or part of a lung, subcrepitant, coarse, or gurgling râles, usually localized, dulness on percussion, and increase in vocal resonance according to the extent of the consolidation.

The fact that the inflammation involves the roots of the lungs rather than their apices causes the physical signs to be heard largely over the central portions of the lungs, while in tubercular inflammation of the lungs it is over the upper lobes that the physical signs are usually heard.

The symptoms continue for weeks or months, the patients gradually lose flesh and strength, and finally die from the interference with breathing or worn out with the disease.

The *Diagnosis* is apt to be difficult. The symptoms resemble those of chronic tuberculosis, of aneurism of the aorta, of intrathoracic tumors, and of actinomycosis of the lung. We are very dependent upon the history of the patient and the presence of other syphilitic lesions.

Treatment.—It is natural in these patients to adopt an energetic treatment with mercury and the iodide of potash, although the rule seems to be that the disease is fatal.

CASE I. *Syphilitic Pneumonia; Death with Profuse Hæmoptysis.*—Male, 60 years old, February 8, 1868.

The man came into the hospital coughing up large quantities of blood, and died in a few hours.

Autopsy.—There is the scar of a chancre on the penis.

The surface of the sternum is eroded..

There is a gummy tumor in the liver.

In the apex of the left lung there are several small cavities full of blood. Around these there is a diffuse growth of fibrous tissue in which are several small, gummy tumors.

In the apex of the right lung there is a diffuse growth of fibrous tissue in which are gummy tumors.

CASE II. *Syphilitic Pneumonia; Diffuse Hepatization and Gummy Tumors.*—Male, 43 years old, March 1, 1883.

Three years ago he began to have cough, pain in the left side, and dyspnœa. Cough and muco-purulent sputum have continued ever since.

Six months ago he had several small hæmoptyses.

Now he is pale and emaciated, the tongue dry and brown, with dyspnœa, cough and profuse muco-purulent sputum; temperature 97° to 99°, pulse 104, breathing 40; urine 1014, a large quantity of albumin.

A week later he died.

Autopsy.—The brain was not examined.

The heart is normal.

The left pleural cavity is half full of purulent serum. The costal and pulmonary pleuræ are coated with fibrin and pus. The lung is compressed and unaërated. In the lower lobe are a number of gummy tumors, from the size of a pea to that of a chestnut, surrounded by diffuse fibrous tissue.

The entire right lung is in the condition of red hepatization. The walls of the air spaces are thickened, without much cell growth. Some of them contain epithelium, fibrin, and pus, others a fibrillated or homogeneous basement substance containing cells and new blood vessels.

The kidneys weigh 24 ounces; the Malpighian tufts are waxy and there are marked changes in the epithelium of the cortex tubes.

CASE III. *Syphilitic Pneumonia; Necrosis of Trachea and Large Bronchi.*—Female, 33 years old, July 20, 1888.

Five months ago she began to have cough with muco-purulent sputum and dyspnœa on exertion.

Six weeks ago she began to have pain in the throat on swallowing.

Ten days ago the voice changed to a stridulous whisper. She has been steadily losing flesh and strength.

Now she is pale and emaciated, the tongue coated and dry, pulse 120, breathing 40, temperature 103°.

There is stridulous breathing, laryngeal dyspnœa, and hardly any voice.

Over both lungs there is sibilant and sonorous breathing with poor pulmonary resonance.

Urine 1020, trace of albumin.

August 3 tracheotomy was performed to relieve the dyspnœa.

October 6 there were flatness on percussion and coarse râles over the whole left chest. The temperature ran from 98° to 104°.

After three months in the hospital she died.

Autopsy.—The brain was not examined.

The heart, liver, spleen, and stomach are normal.

The kidneys are a little enlarged, their capsules adherent, the cortex thick and pale.

The larynx shows no change in the epiglottis or vocal cords, but the laryngeal cartilages are necrotic. The whole length of the trachea and the large bronchi of the left lung are congested, ulcerated, and irregularly thickened and dilated.

The right lung is large, congested, and œdematous.

The left lung is covered with old adhesions. The entire lung is in a condition of smooth, dense hepatization with small bronchiectatic cavities in the lower lobe. There is an extensive diffuse growth of new connective tissue in the walls of the air spaces, the bronchi, and the arteries, and in the interlobular septa, so that a large number of the air spaces are obliterated.

GANGRENE OF THE LUNG.

Definition.—Death accompanied by putrefaction of a portion of the lung.

Etiology.—Whenever the vitality of a portion of the lung is impaired and at the same time the bacteria of putrefaction are present, there may be gangrene. It is found, therefore, with lobar pneumonia, hemorrhagic infarctions, compression or embolism of the pulmonary or bronchial vessels, wounds of the lung, contusions of the chest, cavities in the lung, foreign bodies in the bronchi, cancer of the œsophagus, and in persons whose health has been enfeebled by disease or privation.

This is the ordinary account of gangrene of the lung. In my own experience I have seen gangrene occurring late in lobar pneumonias and as a primary disease.

The primary cases are not uncommon in New York. They occur in persons whose health has previously been good, without discoverable cause. They run an acute course lasting for several weeks, and terminate in death, in recovery, or go on for a long time with cough and expectoration.

Morbid Anatomy.—Gangrene of the lung is either circumscribed or diffuse.

Circumscribed gangrene occurs in the form of one or more foci of small size where the lung is of blackish or greenish color, soft, or broken down into ragged cavities. These foci have a most offensive odor. The lung around them is inflamed and the air spaces contain epithelium, fibrin, and pus. Such gangrenous foci when once formed may increase in size; as they do so the adjoining veins may become filled with infectious thrombi, or eroded. From the thrombi infectious emboli can be carried into the circulation and set up inflammatory foci in different parts of the body. From the eroded vessels there are considerable hemorrhages. If the spot of gangrene is near the pleura it may set up either a simple or a gangrenous pleurisy. Or the pulmonary pleura may be perforated and pyopneumothorax result. Intense bronchitis, either catarrhal or croupous, may be excited by the irritation of the gangrenous matter. If the gangrenous [illegible] gangrenous [illegible] formed of which the walls are [illegible]. Such a cavity may [illegible] tracted.

Diffuse gangrene may be [illegible] or it may be [illegible] lobe, a whole lobe, or even a whole lung, may be involved. The portion of lung involved is changed into a soft, foul-smelling, blackish or greenish mass.

Symptoms.—The patients have a cough with more or less fetid expectoration and a fetid breath. There is much variety as to the quantity of the expectoration; it may be scanty or abundant. When it is abundant and is allowed to stand for a time in a glass it separates into three layers: the upper, frothy, opaque, and of a dirty gray or yellowish color; the middle, clear and watery; the lower, greenish and purulent, or mixed with blood. It consists of serum, mucus, pus, and shreds of lung tissue. There may be, however, only a very fetid breath without any expectoration. If the pulmonary vessels are eroded large quantities of blood are coughed up.

In quite a good many cases there is only muco-purulent sputum with a fetid breath.

The patients have an irregular fever, they lose flesh and strength and pass into the septic condition. But some of the patients who apparently have gangrene of the lung and recover are not at any time so sick as one would expect.

The physical signs are sometimes obscure, sometimes well marked. They are most commonly found over the middle of the chest behind. At this point we may get dulness or flatness on percussion; absence of breathing, bronchial breathing, or cavernous breathing; exaggerated or bronchial voice, and coarse râles.

If the pleura is inflamed or perforated, producing pneumothorax, the symptoms belonging to these conditions are added.

The ordinary cases in New York begin with chills, fever, cough, muco-purulent sputum, pains in the chest, and fetid breath. They go on, with fairly high temperatures, cough and a good deal of sputum, fetid breath, the physical signs of bronchitis, consolidation, or cavities, for several weeks until the disease terminates in death or recovery.

CASE I. *Gangrene of the Lung; Death.*—Male, 47 years old, November 23, 1895.

Seven weeks ago he began to have cough with muco-purulent sputum, offensive breath, and pain in the left side. He has continued to cough, to lose flesh and strength, and to have dyspnœa.

Now, in the eighth week, over the left chest behind, from the spine of the scapula down, there are flatness on percussion, bronchial voice and breathing, and subcrepitant râles.

The man grew weaker and died in three days.

Autopsy.—In the lower lobe of the left lung there is a large, irregular cavity with gangrenous walls surrounded by red hepatization.

CASE II. *Gangrene of the Lung; Death.*—Male, 57 years old, January 5, 1897.

Ten days ago he had a chill, fever, cough, profuse sputum, and pain in the left side of the chest, with nausea and vomiting. A few days ago the breath and sputum became fetid.

Now over the middle of the left lung behind there are dulness on percussion and diminished voice and breathing. Over the rest of the lung there are sibilant and sonorous breathing and coarse râles.

The breath is very foul and he coughs up brown, foul-smelling sputum. His symptoms continued until he died in the tenth week.

Autopsy.—The heart is normal.

The right lung is congested and œdematous, the bronchi are filled with muco-pus.

The left lung is bound down by old adhesions. The middle and lower portions of the upper lobe are converted into a large cavity with necrotic walls infiltrated with foul pus. The rest of the upper lobe is consolidated and studded with small cavities filled with pus. The lower lobe is congested, with small areas of consolidation.

The liver, stomach, and intestines are normal.

The spleen is soft.

The kidneys are in the condition of acute degeneration.

(For Chart of Case II, see Fig. 72.)

CASE III. *Gangrene of the Lung; Recovery.*—Male, 48 years old, November 6, 1896.

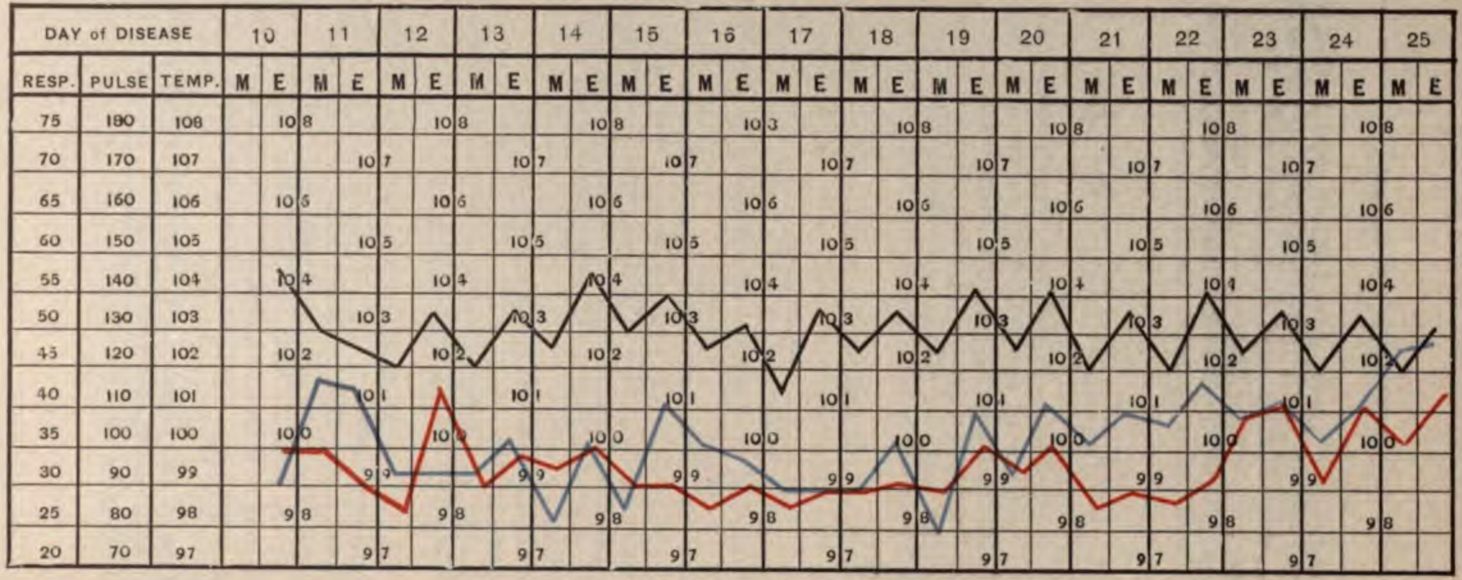
Two weeks ago he began to have cough with profuse, foul, yellow sputum, fever, headache, and pain in the chest.

Now, on the fifteenth day, the color and nutrition are fairly good, the tongue is moist and coated; over the lower part of the left lung behind there are dulness on percussion and subcrepitant râles; urine 1022, no albumin.

After the twenty-third day he began to improve, and was discharged cured on the thirty-sixth day.

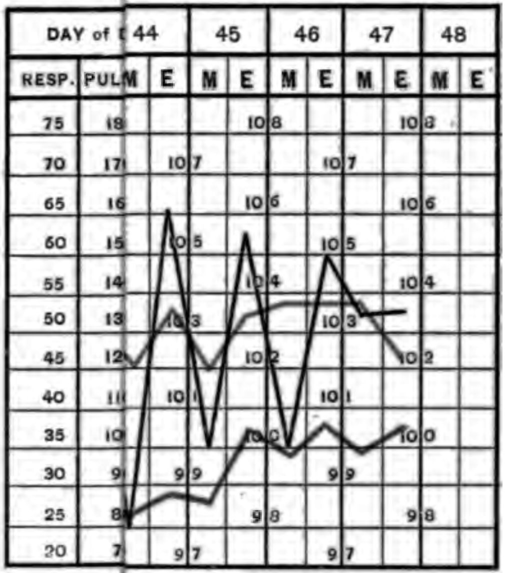
(For Chart of Case III, see Fig. 73.)

FIG. 72



Temperature black, Pulse red, Respiration blue

CASE II.—GANGRENE OF THE LUNG.



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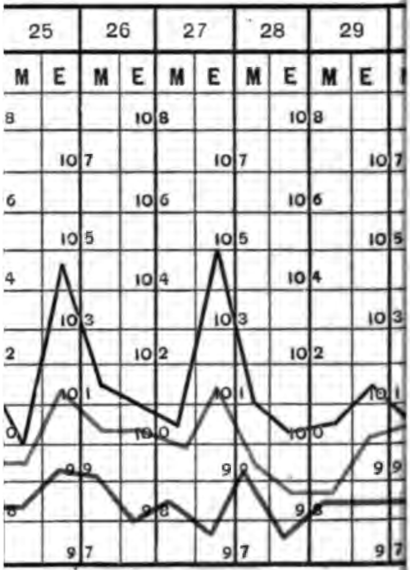

d, Respiration blue

F THE LUNG.

CASE IV. *Gangrene of the Lung; Recovery.*—Male, 38 years old, February 8, 1899.

One week ago, after exposure to cold and wet, he went to bed with chills, fever, and sweating. In the evening he became delirious. He developed cough with profuse, yellow, foul sputum, sometimes mixed with blood.

Now, on the eighth day, his color and nutrition are good, his tongue is coated and dry. In the right interscapular region there are dulness on percussion, bronchial breathing, and coarse râles. From the spine to the angle of the scapula there are dulness on percussion, diminished breathing, and subcrepitant râles.

On the fifteenth day the man began to improve and to get out of bed.

On the sixtieth day he was discharged, without cough or sputum, very nearly well.

(For Chart of Case IV, see Fig. 74.)

CASE V. *Gangrene of the Lung, Protracted.*—Male, 29 years old, November 15, 1899.

One month ago he began to cough. The sputum was at first scanty and mucous, and has gradually become profuse and mucopurulent; it smelt badly from the first. He has lost flesh and strength and for the last two weeks has been in bed.

Now, in the fifth week, he is feeble and emaciated. Over the whole left lung there are sibilant breathing and coarse râles. Over the right lower lobe there are dulness on percussion and subcrepitant râles.

Sixth week, the dulness over the middle and lower portions of the right lung is more marked. There are no tubercle bacilli in the sputum.

The sputum separates into the three layers characteristic of gangrene of the lung; the quantity has reached fifteen ounces for the twenty-four hours.

After this he improved considerably, and the sputum diminished to six ounces for twenty-four hours.

In this condition he left the hospital.

(For Chart of Case V, see Fig. 75.)

The diagnosis is often difficult. It may be evident that the patient has a serious disease and yet impossible for some time to determine its character. Even when it is certain that there is a pulmonary lesion we cannot always tell whether this is gangrene, abscess, or fetid bronchitis.

The *Prognosis* is always serious, but recovery is by no means impossible.

Treatment.—Besides the employment of such measures as add to the comfort of the patients and contribute to their nutrition, it is customary to give creosote or carbolic acid either by inhalation or by the mouth. Perhaps the simplest plan is to use a naso-oral inhaler moistened with equal parts of creosote, alcohol, and chloroform.

In a moderate number of cases gangrenous cavities in the lungs have been opened and drained.

THROMBOSIS OF THE PULMONARY ARTERIES.

There are few morbid conditions concerning which we are so ignorant as we are of thrombosis of the arteries and veins.

Thrombosis of the pulmonary arteries, as we have already seen, complicates some cases of pneumonia. Rarely it occurs without inflammation of the lung, but of these cases there are so few that we can hardly construct a clinical history.

CASE I. *Thrombosis of the Pulmonary Artery; Pleurisy; Pericarditis.*—Female, 50 years old, July 24, 1873.

For two months she has been sick with pain in the left side and dyspnœa.

Now she is feeble and anæmic, with the left pleural cavity full of fluid. July 27 four pints of fluid were aspirated from the left chest.

After the aspiration she improved for a few days; then the chest filled up again with fluid, and on August 15 she suddenly died.

Autopsy.—The brain was not examined.

The pericardium is coated with a thick layer of fibrin.

The left pleural cavity is nearly full of fluid, with the lung compressed.

In the right lung the branches of the pulmonary artery are filled with firm red, white, and yellow thrombi.

The liver shows the changes of chronic congestion.

The kidneys are normal.

CASE II. *Thrombosis of the Pulmonary Artery after Thrombosis of the Femoral Vein.*—Female, 40 years old, September 29, 1875.

She has been a hard-working woman of good habits and fair health.

One week ago the veins of the right leg began to swell and became hard and painful. The swelling of the veins gradually extended up to the abdomen.

Now she is feeble and suffers a good deal of pain. In the right leg the internal saphenous vein and its branches are swollen and indurated down to the knee. The track of the veins is marked by a red blush. The leg is stiff and not easily moved.

October 8, the swelling of the veins had extended up to Poupart's ligament.

October 15, she was feeling better.

October 27, there was no more pain in the leg, the swelling and tenderness of the veins had disappeared, and she began to sit up.

November 1, at 5 in the morning, she awoke with dyspnœa and severe pain in the back; she rapidly became pulseless and died in half an hour.

Autopsy.—The brain is normal.

The heart is normal.

The internal saphenous, femoral, and profunda veins of the right leg are filled with firm thrombi. The thrombi extend up through the iliac veins and into the inferior cava for four inches.

There is a firm thrombus in the trunk of the pulmonary artery, extending into its two large branches. There are no changes in the lungs.

The other viscera are normal.

CASE III. *Thrombosis of the Pulmonary Artery.*—Female, 19 years old, February 15, 1876.

She came into the hospital for too profuse menstruation. The uterus was curetted, and she went out on April 24 in fair condition.

After leaving the hospital she remained weak, with a little cough, leucorrhœa, pain over the heart, nausea and vomiting.

August 18, 1886, she came back to the hospital. She is now fairly nourished, but weak; the uterus is normal, the urine is normal; she complains of a good deal of pain in the precordial region.

September 1, the precordial pain was more severe and paroxysmal, and she also had cough and dyspnœa.

September 4, pulse 140, temperature 101°; she had repeated attacks of precordial pain with rapid heart action and cold extremities. There were no physical signs, except a systolic murmur at the base of the heart.

She continued to have these paroxysms, and died in one on September 11.

Autopsy.—The brain is normal.

The heart is normal.

Over the left upper and lower lobes there is recent fibrin on the pleura, and in these lobes there are scattered areas of red hepatization, and one spot as large as a hen's egg where the lung tissue is broken down and diffluent, but not gangrenous.

A firm, white, dry thrombus completely fills the pulmonary artery where it enters the left lung, and extends through all its branches. The right pulmonary artery is filled with a similar thrombus where it enters the lung, the thrombus extending into the branches of the artery. The trunk of the pulmonary artery before it enters the lung is empty.

There are no thrombi in any of the large veins.

The liver, spleen, kidneys, stomach, intestines, and uterus are normal.

ASTHMA.

Definition.—An affection characterized by paroxysmal dyspnœa, recurring at intervals, generally in the night, the dyspnœa due to a contraction of the bronchi.

The same name of asthma is also frequently employed to designate the paroxysmal dyspnœa caused by disease of the heart and by contraction of the arteries.

Etiology.—Bronchial asthma occurs most frequently in persons who have pulmonary emphysema, but it is by no means rare in persons whose lungs are normal except for the condition of the bronchi.

The causes which produce an attack of asthma may act directly on the mucous membrane of the bronchi, or indirectly on the bronchi through the blood or the nervous system. So we find some persons who never have an attack of asthma except when exposed to a directly exciting cause; while other persons have constantly recurring attacks for long periods of time without any direct cause for each attack.

Among the direct causes we reckon bronchitis, inflammations and obstructions of the nose, climatic influences, dust, vegetable irritants, chemical vapors, and animal emanations.

The ordinary dust floating in the air; the odor or pollen of many plants and grasses; the vapors of pitch, sulphur, or phosphorus; the peculiar smell of dogs, cats, or horses, are familiar examples of direct causes.

The effect of climate in causing asthma is very marked in some persons. This effect does not follow any definite law, but only the idiosyncrasy of the individual. It does not matter whether the locality is warm or cold, wet or dry, low or elevated. For nearly every asthmatic person we can find some one place where he will have little or no asthma.

Of all the causes of asthma, however, bronchitis is the most frequent. In the patients who belong to this class the bronchitis constitutes the important part of the case, for if the attacks of bronchitis can be prevented there is no more asthma.

Of late years much attention has been called to diseases of the nasal passages as a cause of asthma. I think there is no question that they do constitute one of the causes. But it is going a great deal too far to say that they are the only cause.

Among the indirect causes of asthma we enumerate mental emotions, indigestion, hysteria, gout, heredity, and some of the skin diseases. But it must be confessed that we are often unable to say why a previously healthy person should at some particular time, without any exciting cause, begin to have attacks of asthma.

Morbid Anatomy.—As asthma is a functional disease it has no lesions. But in the bodies of old asthmatics we commonly find the morbid changes belonging to pulmonary emphysema and bronchitis.

Symptoms.—A paroxysm of asthma begins with a feeling of oppression or suffocation about the upper part of the chest which obliges the patient to sit up in order to breathe. The feeling of suffocation continues, and the patient brings into play all the muscles of respiration in order to satisfy the hunger for air. The skin becomes livid, the pulse feeble, and the patient's face shows his suffering. If we listen to the chest, we hear over both lungs the sibilant and sonorous breathing caused by the contraction of the bronchi. Such an attack lasts for hours or days. During the most severe attack the patients look as if they might die at any minute, but yet after a time the attack always subsides.

As attacks of asthma are due to a variety of causes, so they present themselves to us under a variety of clinical aspects.

1. There are the persons who never have asthma unless they have an attack of acute bronchitis. In such persons we have to look at the bronchitis as a real disease, while the asthma is only a complication.

2. There are the persons who only have asthma at certain times of the year and in certain localities, the attacks being caused by the inhalation of the pollen or odor of plants. These persons are said to suffer from "hay fever," "rose cold," "autumnal catarrh," etc.

3. There are persons in whom the asthma only constitutes one of the symptoms of pulmonary emphysema. These cases will be described with emphysema.

4. There are other cases in which the asthma occurs by itself as a pure neurosis. In these persons the disease is apt to be very tenacious, the paroxysms recurring again and again, even after considerable intervals of improvement. In the more severe cases the bronchi are somewhat contracted and the breathing labored nearly all the time, while the spasmodic dyspnœa recurs at regular intervals. The patients become worn out by the constant dyspnœa, the face is one of suffering, the chest is bent forward and stooping, the nutrition is impaired, the whole condition is one of chronic invalidism, but yet life is prolonged and death is usually due to some other disease.

Treatment.—The objects of treatment are to cut short the attacks of asthma, and to prevent the recurrence of subsequent attacks.

To cut short an attack of spasmodic asthma we employ such means as will relax the spasmodic contraction of the walls of the bronchi. This can be done in a variety of ways: inhalation of the fumes of stramonium, nitrate of potash, chloroform, ether, or the nitrite of amyl; hypodermic injections of morphine; the preparations of belladonna or chloral hydrate, given by the mouth or the rectum; the use of the drugs which increase the production of mucus from the bronchi, such as lobelia and grindelia robusta.

To prevent the recurrence of the attacks we examine into the condition of the nasal passages to see whether there is disease there which may cause the asthma. We inquire into the history to determine whether the asthma is not caused by bronchitis, or by the pollen or odor of plants.

If the asthma is a pure neurosis a considerable number of patients can find a climate in which they cease to suffer. But the selection of this climate has to be made experimentally by each patient. There is no rule to guide us. Each person has to travel from place to place until they find the particular spot where they cease to have asthma.

For the patients who cannot travel the most efficient treatment seems to be the long-continued administration of the iodide of potash, the systematic inhalation of compressed air, attention to the feeding, disorders of digestion, and to any conditions which impair the general health.

HÆMOPTYSIS.

Blood which is coughed up comes for the most part from the bronchi; less frequently from the pharynx, from eroded vessels in the walls of cavities, or from aneurisms of the pulmonary artery or of the aorta.

Hæmoptyses occur so frequently with the tubercular inflammations of the lungs, and so much less frequently with other morbid conditions, that any hæmoptysis is regarded with a good deal of suspicion.

In tubercular inflammations of the lungs the bleeding is from the mucous membrane of the bronchi. The quantity of blood coughed up may be large or small. The bleeding may last only a few minutes or continue a number of days. The same patient may have only a single hæmoptysis, or several. There seems to be no period in the course of tubercular inflammation of the lungs which is exempt from the liability to bleeding from the mucous membrane of the bronchi. Especially is it to be remembered that either large or small hæmoptyses may precede by a considerable interval of time any rational symptoms or physical signs of tubercular disease.

In the older cases of pulmonary tuberculosis in which cavities have been formed, the vessels in the walls of these cavities may be eroded, with an escape of blood which is large and dangerous.

The very frequency of the association of hæmoptysis with pulmonary tuberculosis makes it important to enumerate the other conditions under which hæmoptysis may occur. The following are the forms of hæmoptysis which occur without pulmonary tuberculosis:

1. A person has an attack of hæmoptysis only lasting for a short time, but during which he raises a considerable quantity of blood. He has no other attacks and does not develop any pulmonary lesions. The bleeding may follow severe muscular exertion, great mental excitement, or occur without discoverable cause.

2. In women hæmoptysis may take the place of menstruation. Flint says he has seen hæmoptysis occurring at regular intervals for four years after the suspension of the menses. But these cases must be looked on with suspicion. A woman may cough up blood on several occasions instead of menstruating, and then go on to have lung disease.

3. Chronic naso-pharyngeal catarrh is sometimes attended with occasional small hæmoptyses.

4. There is a set of women who are hysterical, anæmic, always suffering from some real or fancied ailment, who from time to time cough up a little blood.

Case I.—Female, 12 years old, December, 1901.

She began to menstruate when she was 10 years old, and her health has been good until within the last year.

During the last year she has had frequent headaches, three attacks of syncope, and some hysterical symptoms. She has coughed up small quantities of blood some twenty or thirty times in the course of the year. Otherwise she has had no pulmonary symptoms and no physical signs.

The hæmoglobin is 55 per cent, the red cells 4,028,000.

5. It is said that pregnant and nursing women sometimes have hæmoptysis.

6. With disease of the aortic and mitral valves, especially with mitral stenosis, bleeding from the bronchi is of frequent occurrence. In the course of the heart disease the patients from time to time, during periods of several days, cough up clear blood in considerable quantities.

7. Aneurisms of the branches of the pulmonary artery within the lungs, when they rupture, cause fatal hemorrhage, a large part of the blood being coughed up.

Aneurisms of the arch of the aorta which erode the trachea or main bronchi may rupture into the trachea or bronchi by small or large openings. With the small openings the patients cough up a little blood from time to time, while part of the escaped blood is inspired into the lungs and sets up a peculiar form of pneumonia. With the large openings the blood escapes through the trachea in enormous quantities, and the patients bleed to death within a few minutes.

8. Patients who suffer from emphysema and chronic bronchitis not infrequently from time to time cough up small quantities of blood. Much less often such patients have at some time a large bleeding from the bronchi. Part of this blood is coughed up at once, part is coagulated in the large bronchi and is afterward coughed up in the form of casts of these tubes.

9. Sir Andrew Clark (*Lancet*, 1889, p. 841) describes a form of bleeding which he calls "arthritis hæmoptysis," of which he says that he has seen some twenty cases. He lays down the following propositions: There occurs in elderly persons, free from ordinary diseases of the heart and lungs, a form of hæmoptysis arising out of minute structural alterations in the terminal blood vessels of the lung.

These vascular alterations occur in persons of the arthritic diathesis, resemble the vascular alterations found in osteo-arthritic articulations, and are themselves of an arthritic nature.

Although sometimes leading to a fatal issue, this variety of hæmoptysis usually subsides without the supervention of any coarse anatomical lesion of either the heart or the lungs.

This variety of hemorrhage is aggravated or maintained by the frequent administration of large doses of strong astringents, and by an unrestricted indulgence in liquids to allay the thirst which the liquids themselves create.

The treatment which appears to be the most successful in this variety of hæmoptysis consists in diet and quiet, in the restricted use of liquids, and the stilling of cough; in calomel and salines, in the use of alkalies, with iodide of potassium, and in frequently renewed counter-irritation.

Case II.—Female, 70 years old, April 25, 1883.

While in her ordinary health she had suddenly coughed up a pint of blood. An hour later she was somewhat prostrated, but without much dyspnœa. There was complete flatness and absence of breathing over the whole left chest.

The next day she was quiet in bed without much dyspnœa.

April 30 she was quiet in bed, no dyspnœa, complete flatness and absence of breathing over whole left chest; the heart was not displaced; there was a systolic murmur over the whole precordial region. That evening she coughed up a clot of blood forming a cast of a principal bronchus and its branches.

May 1, she did not feel as well; no dyspnœa; dulness instead of flatness over the left lung, with feeble breathing.

May 2, she felt better. She coughed up blood casts of the smaller bronchi. The dulness was less and the breathing was louder over the left. In a few days she was entirely well and had no trouble afterward.

10. Dr. Flint ("Practice of Medicine," p. 265) says that he has met with a few cases of persistent bronchial hemorrhage. In two of these cases, after expectoration daily more or less of a sero-sanguinolent liquid during several months, recovery took place under the use of tonics and hygienic measures. In the third case the hemorrhagic expectoration continued for six years, during which time repeated examinations of the chest failed to discover any positive signs of pulmonary disease.

11. Severe injuries inflicted upon the wall of the thorax may be followed by the expectoration of blood for hours or days.

Treatment.—In managing cases of bleeding from the mucous membrane of the bronchi it is important to bear in mind that even the most profuse hemorrhages are seldom fatal.

The methods of treatment commonly employed are: the application of cold to the chest, the temporary ligation of one of the arms or legs, the internal use of opium, ergot, hydrastis, kramaria, tannic acid, gallic acid, acetate of lead, persulphate or pernitrate of iron, or of calomel or of the saline cathartics. It is also customary to keep the patients very quiet while the bleeding is going on. I doubt if it be wise to be too anxious and energetic in the treatment of bleeding from the mucous membrane of the bronchi. The bleeding will regularly stop, no matter what is done. The frequent use of astringents disorders the stomach; the insistence of absolute quiet demoralizes the patients; the keeping the patients on a low diet unnecessarily reduces their strength.

EMPHYSEMA.

INTERLOBULAR EMPHYSEMA.

Definition.—An accumulation of air in the connective-tissue septa between the lobules of the lung.

Etiology.—Interlobular emphysema is, I think, most frequently seen with the broncho-pneumonia of young children. It may be caused by any violent efforts which require the abrupt introduction of a large quantity of air into the lungs, and its forcible retention therein by closure of the glottis. The efforts in parturition, defecation, raising weights, coughing, paroxysms of rage, excessive laughter, and hysterical convulsions have all been occasionally followed by rupture of the air cells and interlobular emphysema.

Morbid Anatomy.—We find after death the interlobular septa infiltrated with air, with more or less compression of the parenchyma of the lung. The air may find its way from the lung into the mediastinum and thence into the connective tissue of the neck and the wall of the thorax.

Symptoms.—There seem to be no distinctive rational or physical symptoms belonging to interlobular emphysema. But in extreme cases it is said to have caused sudden death.

VESICULAR EMPHYSEMA.

It is customary to speak of three forms of vesicular emphysema: Compensating emphysema, senile emphysema, and substantive emphysema.

1. *Compensating Emphysema.*—If one lung, or a part of one lung, is so changed by disease that it can only partially perform

its functions, the other lung becomes increased in size and its air spaces are dilated. This change in the lung is a beneficial rather than a morbid one, and gives no symptoms of disease.

2. *Senile Emphysema.*—This condition is often described as something different from substantive emphysema. It is said that, instead of there being an abnormal accumulation of air in the lungs, these organs are smaller and contain less air than normal. In consequence of atrophy of the alveolar walls the air cells coalesce and form larger air spaces. These, however, do not result from a dilatation of the alveoli, but from a gradual shrinkage and disappearance of the lung tissue.

I must confess that such descriptions of senile emphysema do not correspond with the lungs which I have seen. I believe that the anatomical conditions are the same in senile as in substantive emphysema, although the causation and clinical history are different.

3. *Substantive Emphysema.*—The definition of substantive emphysema usually given is that it is a morbid condition of the lungs characterized by enlarged capacity of the air cells, with atrophy of their walls and obliteration of their capillaries.

Laennec, who was the first to describe this lesion, gives the causation of emphysema as follows: Chronic catarrh, plugging by mucus of small bronchi, consequent obstruction to the passage of air, conveyance by inspiration of air past the obstruction, failure of expiration to expel this air, accumulation of air in the air spaces, dilatation of the air spaces. The foundation of the disease, therefore, is bronchitis.

Louis denies this mechanism, because the symptoms of dilated air cells are not preceded by catarrh, because habitual dyspnœa does not undergo permanent increase after acute catarrh, and because normal inspiration is not more powerful than expiration.

Dr. Williams maintains the catarrhal basis of Laennec, but supposes that, while the air cells communicating with plugged bronchi escape distension, those adjoining and possessed of free communication with the trachea dilate in consequence of the extra work and pressure thrown upon them.

Walshe says that the vesicular dilatation may be a primary or a secondary phenomenon; that is, it may occur independently of any acknowledged form of statical change within the chest, or it may supervene on some actual organic mischief. The dilatation may be the resultant of primary nutritive change in the actual walls of the enlarged vesicles, affecting both their statical and dynamic properties. Or these cells, being in their own nature healthy, may dilate through the extra strain thrown on them in consequence of the inaction of neighboring portions of lung.

It is frequently stated that playing on wind instruments or glass blowing causes emphysema.

I think that it is curious that a real disease of the lungs, such as emphysema is, should not yet have been put in its proper place. It belongs to the chronic productive inflammations of the viscera—arteritis, nephritis, cirrhosis, etc.—and follows the laws which govern them. There are all the degrees, from the lesion that gives no symptoms to the lesion that causes death.

In the autopsy room, if to the naked eye the air spaces are seen to be dilated, the patient is said to have had emphysema. Clinically certain physical signs are said to denote emphysema. In either case the condition is passed over as not of much consequence. And yet, really, emphysema may be a most serious disease, causing marked symptoms and death.

If we once get rid of the idea that emphysema is only dilatation of the air spaces, and think of it as a chronic interstitial pneumonia, the clinical symptoms and the lung lesions will be found to correspond.

Just as in cirrhosis of the liver the two cardinal symptoms are loss of nutrition and dropsy, so in emphysema the cardinal symptoms are loss of nutrition, dyspnœa, and venous congestion of other parts of the body.

Morbid Anatomy.—Both lungs are, as a rule, increased in size. The dilatation of the air spaces may be so great as to be evident to the naked eye, or so moderate as not to be appreciable. When we examine minutely the lungs of a large number of persons who have, during life, suffered from the symptoms of emphysema, we find a very great variety in the morbid conditions. In some both the air passages and air vesicles are largely dilated; in some the air passages alone are dilated, the air vesicles remaining of normal size; in some neither the air passages nor the vesicles are appreciably dilated. We find also, when we compare the lungs with the clinical histories which belong to them, that the most marked symptoms are often associated with very slight degrees of dilatation of the air spaces, and it is evident that the severity of an emphysema is not to be measured by the degree of dilatation of the air spaces.

The walls of the air spaces are thickened in some parts of the lung, thinned in others. The epithelial cells which line the walls of the air spaces are often increased in size and number. In the walls of the air spaces are holes with sharp-cut edges. These holes are formed in the spaces between the capillaries; some are very minute, others are of large size. It is evident that these holes are not formed by the stretching of the air spaces, for they are found in small air spaces as well as in large ones. They constitute a curious and important part of the lesion. The septa between the lobules, the connective tissue around the bronchi and blood vessels, and the pulmonary pleura are often considerably thickened. Very frequently there are adhesions between the pulmonary and costal pleuræ. The mucous membrane of the larger bronchi is often thickened.

The blood vessels of these lungs can be readily filled with an artificial injection. Neither arteries, capillaries, nor veins are obstructed or obliterated; but in the walls of dilated air spaces, and in the walls of those in which there are holes, the meshes of the capillary plexus are larger and the capillaries are farther apart from each other.

During life, however, in some cases of emphysema, there is an obstruction to the passage of blood through the lungs, and consequently, dilatation and hypertrophy of the right ventricle of the heart and venous congestion of the pia mater, stomach, small intestine, liver, spleen, and kidneys. These evidences of venous congestion then exist in cases in which the air spaces are but very little dilated.

In many cases we also find chronic endarteritis, endocarditis, or nephritis.

Symptoms. Physical Signs.—In the lesser degrees of emphysema there is no change in the shape of the thorax. In the more advanced cases there is a prominence of the upper part of the sternum and of the costal cartilages. In patients who have suffered much from dyspnœa the hypertrophy of the muscles which move the thorax contrasts with the general emaciation of the patients. In the cases in which there is great dilatation of the air spaces the chest assumes the so-called barrel shape.

The percussion sound may remain unchanged for a considerable length of time. When it is changed, the change is either to a rather dull note of wooden quality or to exaggerated resonance of either vesicular or vesiculo-tympanitic quality. The respiratory murmur is feeble, or there is feeble inspiration with longer, louder, low-pitched expiration; or both inspiration and expiration may be exaggerated, loud, and high-pitched.

The pleuritic adhesions give more or less dulness on percussion. When the bronchi are contracted there is sibilant and sonorous breathing.

Rational Symptoms.—There are many persons in whom substantive emphysema is developed and continues for many years without giving rise to any symptoms, and yet even in such persons it is often possible to be pretty sure of the presence of the disease, because they are persons whose general condition and age are such as are usually associated with emphysema.

There are many persons in whom the associated chronic endocarditis, or endarteritis, or nephritis gives such marked symptoms that the emphysema passes unnoticed.

In some persons the emphysema produces after a time dyspnœa on exertion, but without bronchitis or disturbance of the general health. These are persons past middle age, who do not consider themselves invalids, who, on the contrary, are often strong and robust, and in whom the emphysema is only an inconvenience.

In some persons the principal symptoms are those belonging to the associated acute and chronic bronchitis. The attacks of acute bronchitis may be mild, lasting for a few days or a few weeks, with cough, mucous expectoration, sometimes hæmoptyses, asthmatic breathing, and a febrile movement; or they may be severe and last for several months, with continued cough, asthmatic breathing, fever, venous congestion, dropsy, and loss of flesh and strength. The chronic bronchitis continues year after year, better every summer and worse every winter. The patients have a cough, with mucous or muco-purulent expectoration, sometimes with small hæmoptyses. They are always a little short of breath when they exert themselves. After a time they have attacks of spasmodic asthma. In the unfavorable cases the dyspnœa on exertion becomes more constant and more decided, venous congestion and dropsy are established, and the patients lose flesh and strength.

In some persons the prominent feature in the disease is the liability to attacks of spasmodic asthma, which often are frequently repeated and of long duration. These attacks are sometimes due to contractions of the bronchi, and then we get sibilant and sonorous breathing; or they are due to contraction of the arteries belonging to the aortic system, and there is a radial pulse of increased tension; or they are due to contraction of the branches of the pulmonary artery, and then there is neither sibilant and sonorous breathing nor a pulse of high tension.

In some persons the principal symptom is the constant dyspnœa. The difficult breathing is at first only developed by exertion; later it becomes more constant and is made worse by slight exertion, by indigestion, and by bronchitis. Finally, in the bad cases, the dyspnœa is constant and distressing. The patients constantly feel the need of air, and are always overusing the muscles of respiration in order to satisfy this need. General venous congestion is gradually established, as well as cyanosis of the skin, clubbing of the fingers, congestion of the stomach, small intestine, liver, and kidneys, dilatation and hypertrophy of the right ventricle of the heart, and general dropsy. The nutrition of the patients suffers and they become emaciated, feeble, and anæmic. It is not easy to tell how much of this dyspnœa depends upon the anatomical changes in the lungs, how much upon contraction of the branches of the pulmonary artery, and how much upon contraction of the arteries belonging to the aortic system.

To give an idea of the cases in which emphysema is the primary lesion, I give a table of eight cases. In these it will be seen that the emphysema is the one lesion that caused the symptoms, the secondary lesions, and the death of the patient.

Case.	Age.	Sex.	Lungs.	Heart.	Kidneys.	Liver.	Brain.	Symptoms.
1	65	♂	Emphysema. Bronchitis. Old adhesions. Diffuse black connective tissue in both upper lobes.	Right ventricle hypertrophied.	Normal..	Congested.	Pia thickened. Serum increased.	Three months dyspnœa and prostration. Two weeks œdema of legs and dry cough. Urine 1020, no albumin.
2	56	♂	Emphysema. Bronchitis. Bands and nodules of fibrous tissue. Old adhesions.	Right ventricle dilated and hypertrophied.	Chronic congestion.	Large, nutmeg.	Normal..	Two years short of breath. Barrel chest. Emphysematous breathing, dry râles. Cyanosis, delirium, dyspnœa.
3	36	♀	Marked emphysema. Muco-pus in bronchi.	Right auricle dilated. Right ventricle hypertrophied.	Medium size. Hard and congested. Capsules adherent.	Normal.	Normal..	Two weeks before death, cough, constant dyspnœa, œdema of legs. Died with dyspnœa, feeble and irregular heart.
4	42	♂	Emphysema. Bronchitis with muco-pus. Intense œdema and congestion of the lungs.	Old pericardial adhesions.	Normal..	Nutmeg.	Not examined.	Two years emphysema and bronchitis. Sudden attack of œdema of the lungs.
5	44	♀	Emphysema. Bronchitis. Fibrous tissue at right apex.	Right ventricle dilated and hypertrophied.	Chronic congestion.	Small, hard, finely nodular.	Not examined.	Six years cough, muco-purulent sputum, hæmoptyses. Two years asthma. Five months worse, with cough, dyspnœa, cyanosis, emaciation. Died with dyspnœa.
6	48	♂	Emphysema. Bronchitis. Old adhesions. Congestion and œdema.	Right ventricle hypertrophied. Left ventricle small.	Combined weight sixteen ounces. Chronic congestion.	Hard and congested.	Not examined.	Twenty-four years winter cough. Five years dyspnœa. Three months constant cough. Now bad dyspnœa, cyanosis, œdema of feet, urine trace of albumin, hyaline and granular casts. Died with dyspnœa.

Case.	Age.	Sex.	Lungs.	Heart.	Kidneys.	Liver.	Brain.	Symptoms.
7	24	♂	Emphysema. Bronchi congested. Congestion of lower lobes.	Normal	Normal..	Normal.	Not examined.	Note vesiculo-tympanitic, expiration prolonged, and mucous and subcrepitant râles. For few days before death fever and great dyspnœa.
8	73	♂	Emphysema. Bronchitis. Old adhesions of right lung. Left pleura one-third full of serum. Congestion and œdema.	Right ventricle dilated and hypertrophied. Left ventricle dilated. Aortic and mitral valves a little thick.	Congested.	Congested.	Not examined.	Fifteen years winter cough. Six weeks ago cough, with constant dyspnœa, worse in attacks. Diminished resonance, prolonged expiration. Heart irregular and feeble. Urine trace of albumin. Died with dyspnœa, œdema of lungs and legs.

There are rare and fatal cases in which there are no pulmonary symptoms. The patients lose flesh and strength, and become anæmic without any evident cause for their ill health. After continuing in this way for some time, they begin to have attacks of contraction of the arteries, with headaches, sleeplessness, delirium, stupor, muscular twitchings, and vomiting, or a dyspnœa like that seen with chronic nephritis. They die within a few months after they have begun to have the attacks of contraction of the arteries.

In some persons, after emphysema has existed for some time, with more or less marked symptoms, chronic miliary tuberculosis is slowly established.

Emphysema by itself proves fatal in a moderate number of cases. Death is usually due to some complicating or intercurrent disease.

Treatment.—The conditions which call for treatment are the morbid condition of the lung, the loss of nutrition, the bronchitis, the constant dyspnœa and the spasmodic dyspnœa, the contraction of the arteries, and the venous congestion.

The emphysema is favorably affected by an out-of-door life in a suitable climate; by abstinence from alcohol and tobacco; by the use of fats and by overfeeding with the stomach tube; and by methodical inhalations of compressed air. All of these measures are also of service in improving the nutrition of the patients and in helping them to get rid of chronic bronchitis.

The constant dyspnœa is due to the changes in the lungs, and is then to be treated by the same means which are used to control the emphysema; or it is due to the complicating contraction of the arteries, and is then to be treated by the drugs which dilate the arteries—nitroglycerin, potassium iodide, or chloral hydrate.

The attacks of spasmodic dyspnœa are due to:

(*a*) Spasmodic contraction of the muscular coat of the bronchi. This can be relieved by the inhalation of the fumes of stramonium, nitrate of potash, chloroform, or ether, by the administration of chloral hydrate, potassium iodide, belladonna, or opium.

(*b*) Congestion of the walls of the bronchi. This can be relieved by drugs which increase the production of mucus, such as lobelia and grindelia robusta; by drugs which stimulate the heart, such as caffeine, convallaria, and digitalis; or by the application of dry cups to the walls of the chest.

(*c*) Contraction of the small arteries. This can be relieved by the drugs which dilate the arteries, such as nitrite of amyl, nitroglycerin, chloral hydrate, potassium iodide, or opium.

(*d*) Inflammation of the nasal passages. This is to be treated by local applications.

ACTINOMYCOSIS OF THE LUNG.

The following account of actinomycosis of the lung is taken from a compilation of thirty-four cases made by Dr. Hodenpyl; my own experience is limited to two cases.

Definition.—Pulmonary actinomycosis is a chronic infectious disease of the lungs dependent upon the presence of actinomyces.

Etiology.—The living germ seems to be inhaled into the bronchi. The majority of the cases were in young adults; the youngest patient was 9 years old, the oldest 63.

Morbid Anatomy.—The lesions are unilateral in about the proportion of three to one. They may be classified into two groups:

1. There are cases with the symptoms of chronic general bronchitis, with the germ present in the sputum, but in which no autopsy is made. Whether in these cases there is no lesion but that of chronic bronchitis we cannot certainly tell.

2. There is broncho-pneumonia of a peculiar type, which involves part of a lobe or an entire lung. The largest bronchi are coated with muco-pus. The small bronchi contain pus, their walls are thickened, they are surrounded by zones of peribronchial pneumonia. In some of the small bronchi there are growths of new connective tissue partly filling them. In the peribronchial zones of pneumonia the walls of the air spaces are thickened and their cavities filled with new connective tissue. Between these zones is a diffuse hepatization of ordinary exudative type. There are adhesions between the pulmonary and costal pleuræ. There are often, in addition, sacculated collections of pus in the pleural cavity, which may perforate through the skin or through the diaphragm. The ribs, sternum, or vertebræ may be eroded. The opposite lung, the pericardium, or the heart may become involved. There may be secondary inflammations of the abdominal organs or of the brain. In one case the inflammation penetrated the portal vein and there were metastases all through the body.

Symptoms.—A febrile movement is present in nearly all the cases. Usually it is one of the first symptoms, but sometimes it does not come on until later in the course of the disease.

Cough is usually the first symptom and continues throughout the disease. It is accompanied with an abundant muco-purulent, often fetid, expectoration, and sometimes contains actinomyces. Hæmoptyses were not observed, although the sputa were sometimes stained with blood.

The patients lose flesh and strength, at first slowly; later, as abscesses are formed and septic poisoning established, they run down more rapidly.

The physical signs are those of bronchitis, of broncho-pneumonia, of phthisis, or of empyema.

The average duration of the disease is ten months; the shortest case lasted four months, and the longest was still living after a duration of several years.

Of the thirty-four cases, all died except two.

Diagnosis.—The disease is liable to be confounded with fetid bronchitis, gangrene of the lung, broncho-pneumonia, or pulmonary phthisis. The only positive diagnostic symptom seems to be the presence of the actinomyces in the sputa or in the pus from the pleura.

Treatment.—There seems to be no way of directly improving this disease of the lungs.

It is said, however, that good results have been obtained by the use of iodide of potash in large doses.

THE MALIGNANT GROWTHS OF THE LUNGS.

Carcinomata of the lung occur as primary and secondary growths. The secondary growths follow the anatomical type of the primary tumor.

The primary growths consist of a stroma enclosing spaces lined with cylindrical epithelium, the growth apparently beginning in the small bronchi. Whether the tumors are primary or secondary, we find that they may be developed in such a way as to compress the bronchi; or be associated with exudative and productive inflammation in such a way as to consolidate large portions of the lung; or be associated with suppurative and destructive inflammation in such a way that abscesses are formed; or involve the pleura, so that collections of serum are formed in the pleural cavities.

The sarcomata of the lungs are secondary tumors. They usually are in the form of nodules scattered through the lung, or of tumors which compress the bronchi, or of tumors in the pleura with serum in the pleural cavities.

The lymphomata begin in the bronchial glands and infiltrate the lungs from the root outward, following the track of the bronchi or of the interlobular septa.

Symptoms.—While in some cases the symptoms are obscure, in others they are well defined. They are apt to follow one of three types:

1. The most marked symptom is dyspnœa, due to pressure on the bronchi. The dyspnœa is developed slowly and is brought on by exertion. It becomes more and more distressing, until finally the patient can hardly move at all without bringing on the difficulty in breathing.

The physical signs are either tubular breathing or diminished breathing over the lung corresponding to the compressed bronchi. The patients lose flesh and strength, at first slowly, later very rapidly.

2. The symptoms are those of a chronic inflammation of the lung. The patients have cough, dyspnœa, muco-purulent or bloody expectoration, a febrile movement and pain in the chest, the physical signs of bronchitis and of consolidation of the lungs, and gradual loss of flesh and strength.

3. The symptoms are those of a pleurisy with effusion, but it is a pleurisy which does not improve under treatment. The serum is apt to be blood-stained, but is not always so; it reaccumulates after it has been drawn off: the patients steadily lose flesh and strength.

My own experience with secondary carcinomata of the lung has been principally with those which occur after removal of cancer of the breast. It has seemed to me that since surgeons have practised the very extensive removal of tissue in operating on cancer of the breast, we see fewer recurrences in the scar and more recurrences in the lungs and liver. These recurrences come late—two or three years after the operation.

Some time, then, after the operation these patients begin to have dyspnœa on exertion, with or without cough, but there are no physical signs. The dyspnœa is not at first very urgent, and the patients are not at all ill. But as the months go on the dyspnœa is more and more easily brought on by less and less exertion, the cough is more troublesome, and the flesh and strength begin to fail. But even at this time there may be intervals of considerable improvement lasting for several weeks. Then, after a time, come the physical signs of fluid in the chest, of bronchitis, of pressure on the large bronchi, or of consolidation.

In the primary cases the first symptoms have been the dyspnœa and cough. The curious thing about the dyspnœa is that, while in all cases it is at first a dyspnœa on exertion and then an uncontrollable, constant dyspnœa, its character is the same whether the growth is in the pleura, the lung, or around the bronchi.

The physical signs are those of fluid in the pleural cavity, of bronchitis, of narrowing of the bronchi, or of consolidation of the lung.

CASE I.—Female, 54 years old.

In September, 1877, she began to have dyspnœa on exertion, followed by a dry cough. Later there was profuse mucous and bloody sputum and constant dyspnœa.

In January, 1878, there were the physical signs of a general bronchitis and of fluid in the left pleura.

January 21 she died.

Autopsy.—There is serum in both pleural cavities.

The lungs are large, firm, and studded within and without with small white nodules. The bronchial glands are large, hard, and infiltrated. The nodules in the lungs are formed of air vesicles filled with large, irregular cylindrical cells and of new connective tissue infiltrated with similar cells.

The other viscera are normal.

CASE II.—Male, 43 years old.

In August, 1877, he began to cough with mucous sputum and to have dyspnœa. He was treated for tubercular laryngitis.

The dyspnœa gradually increased and he had constant pain in the left side. Lately he has lost flesh and strength and has had night sweats.

January 15, 1878, I first saw him. He then had the physical signs of fluid in the left chest. I looked upon the case as one of pleurisy with effusion.

February 14 I aspirated four pints of clear yellow serum.

February 27 I aspirated five pints of clear serum. The temperature ran from 99° to 101°, the dyspnœa grew steadily worse in spite of the aspiration, he continued to lose flesh and strength, and died on March 13.

Autopsy.—The larynx, trachea, and large bronchi are congested and coated with mucus.

The left pleural cavity is nearly full of clear serum; the pleura is studded with small white nodules.

The lower lobe of the left lung is converted into a solid, uniform white mass. The upper lobe is studded with small white nodules. The whole right lung is studded with small white nodules.

The new growth consists of air spaces filled with cylindrical cells, and of a diffuse new connective tissue containing alveoli filled with large round cells. In the left lower lobe the consolidation is due partly to new growth, partly to exudate in the air spaces.

The pericardial sac is distended with turbid serum.

The liver is large and contains a number of small white nodules.

The other viscera are normal.

Case III.—Female, 27 years old, January 22, 1882.

Six months ago she began to have cough with mucous and bloody sputum.

For two months she has been confined to bed with night sweats, irregular chills, dyspnœa, nausea and vomiting, pain in the right side, and loss of flesh and strength.

Now there is marked tracheal dyspnœa—constant, with exacerbations—sonorous and sibilant râles over both lungs. I thought she had a small aneurism pressing on the trachea. She continued in the same condition with constant dyspnœa, temperature 96° to 102°.

February 13 she died.

Autopsy.—The larynx and the upper part of the trachea are normal.

The wall of the lower end of the trachea is thickened, its inner surface is irregular and papillary. The epithelial cells are increased in size and number, and in places the cylindrical cells are replaced by flat cells. The wall is thickened from the basement membrane back to the cartilage by a new growth of connective tissue. Although the wall of this part of the trachea is thickened, its lumen is not changed.

The walls of the large bronchi between the trachea and the lungs are very much thickened, and the lumen narrowed so that a goose-quill will hardly pass through the largest bronchus. The epithelium is thickened, the cells deformed, and there are patches of pavement epithelium. The stroma is thrown into papillæ of irregular size and shape. The thickening of the walls of the bronchi is made up of connective tissue.

The walls of the medium-sized bronchi within the lungs are moderately thickened.

Through the lungs are patches of hepatization and quite a good deal of interstitial pneumonia.

There are firm white clots in the pulmonary arteries.

The heart, aorta, liver, stomach, intestines, spleen, kidneys, and uterus show no morbid changes.

LECTURES

ON

THE PRACTICE OF MEDICINE

WITH CASES AND CHARTS

BY

FRANCIS DELAFIELD, M.D., LL.D. (Yale)

PART III

THE HEART

FOR SALE BY J. T. DOUGHERTY
409 & 411 West 59th Street
Branch: 334 East 26th Street
NEW YORK

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THE HEART.

In examining any person who is suspected of having a disease of the heart we follow a certain routine. We determine the size of the heart, the character of its impulse, the rhythm of its contractions and dilatations, the character of the heart sounds, and the presence or absence of murmurs. Most important of all, we try to tell how well the heart is carrying on the circulation of the blood.

The position of the heart and of its valves is seen in Fig. 76. This drawing has been made from dissections in the department of anatomy of the College of Physicians and Surgeons. It accurately represents the average position of the heart.

The size of the heart is made out by simple or auscultatory percussion and by locating the apex beat. It is usually easy to make out an increase in the size of the heart, but a diminution in its size is much more difficult to determine.

The apex beat can usually be felt by the hand, but in many healthy persons can hardly be appreciated. Its position must then be determined by the stethoscope. The force of the impulse is temporarily increased by muscular exertion, by rapid respiration, by digestion, and by mental emotions. It is decreased by all conditions that weaken the heart, such as dilatation of the ventricles, myocarditis, endocarditis, fluid in the pericardium, and general loss of nutrition. But many persons whose health is good have habitually a feeble heart impulse.

The force of the impulse is increased in some of the cardiac neuroses, with hypertrophy of the ventricles, with contraction of the arteries, and with some of the inflammations of the pericardium and endocardium.

Dilatation of the ventricles changes the character of the impulse; instead of a circumscribed apex beat, there is a diffuse, heaving impulse.

The rhythm of the alternate contractions and dilatations of the heart should be perfectly regular. In some healthy persons, however, there is a regular intermission of the ventricular systole. Intermissions also occur in the tubercular meningitis of children.

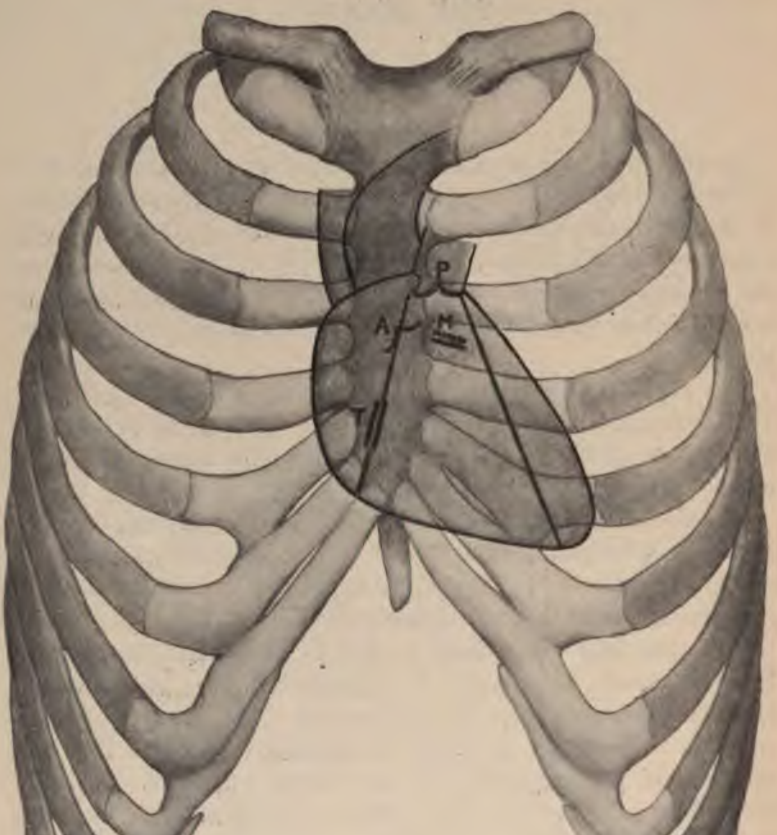

FIG. 76.—*A*, aortic valve; *P.* pulmonary valve; *M* mitral valve; *T.* tricuspid valve.

The heart's action becomes irregular with valvular disease, dilatation and hypertrophy of the ventricles, fatty degeneration of the wall of the heart, myocarditis, pericarditis, and the cardiac neuroses.

The contraction and dilatation of the ventricles are accompanied with certain sounds. The first sound is synchronous with the systole of the ventricles, the apex beat, and the closure of the auriculo-ventricular valves: it is loudest at the apex. After the first sound is a short interval of silence—the post-systolic silence. The second sound is synchronous with the closure of the aortic and pulmonary valves: it is most distinct over the third cartilage and the middle of the sternum. After the second sound there is another interval of silence—the post-diastolic silence.

When we listen to the heart sounds we notice the loudness, the distinctness, and the quality.

Murmurs are pericardial or endocardial.

Pericardial murmurs are produced by the rubbing of opposed surfaces of pericardium covered with fibrin or connective tissue.

Endocardial murmurs are produced by:

(*a*) Changes in the valves by which they are rendered rough, stenosed, or insufficient.

(*b*) Ventricular lesions. Inflammation of the endocardium, or of the chordæ tendinæ; abnormal tendinous cords extending across the ventricles, or thrombi in the ventricles. Ventricular murmurs are systolic; their point of maximum intensity is at the apex.

(*c*) Systolic murmurs heard at the second left interspace, or at the apex without disease of the heart or pericardium.

PERICARDITIS.

Lesions.—The inflammations of the pericardium resemble those of the pleura.

They begin acutely or subacutely, and then run an acute, subacute, or chronic course. There is a greater disposition to bleeding from the inflamed surfaces than is the case in pleurisy. The inflammation regularly begins at the base of the heart and then extends over the greater part of the pericardium.

We distinguish:

1. *Pericarditis with the Production of Fibrin.*—The pericardium is congested, or even studded with minute hemorrhages. Its surface is coated with a thin film of fibrin, or with large, rough masses which glue together the opposed surfaces of the pericardium. The quantity of fibrin is sometimes large, and may be so distributed that during life it gives the effect of enlargement of the heart or of fluid in the pericardial sac. If the inflammation is protracted the pericardium itself becomes thickened and infiltrated with cells, and the wall of the heart may also undergo inflammatory changes.

If the patient recovers the fibrin is absorbed and the pericardium returns to its normal condition; or there is a growth of new connective tissue, which forms permanent thickenings and adhesions.

2. *Pericarditis with the Production of Fibrin and Serum.*—The pericardium is coated with fibrin and there is an effusion of serum in the pericardial sac. The serum accumulates at first between the floor of the pericardium and the lower surface of the heart; as it increases it distends the pericardial sac in all directions, pushing the heart upward and forward. The pericardial sac may be so much distended as to compress the trachea, the left bronchus, the œsophagus, or the aorta. If the patients recover, the serum and fibrin are absorbed, but permanent thickenings and adhesions of connective tissue are left.

3. *Pericarditis with the Production of Fibrin, Serum, and Pus.*—This variety may have the purulent character from the outset, or it may begin as one of the forms just described and afterward assume the purulent character. The inflammation is apt to run a chronic course. The pericardial sac contains a large quantity of purulent serum; the pericardium is coated with fibrin, thickened and infiltrated with cells; the walls of the heart may be the seat of interstitial myocarditis. The trachea, left bronchus, œsophagus, or aorta may be compressed. The serum may be absorbed, and the fibrin and pus undergo cheesy degeneration, or

there may be an infiltration of the thickened pericardium with the salts of lime.

Causes.—Inflammation of the pericardium is caused by pneumococci, streptococci, staphylococci, the bacillus of influenza, the bacillus of typhoid fever, the tubercle bacillus, the poisons of rheumatism, of scarlatina, of smallpox, and of some of the other infectious diseases, and it occurs in the course of chronic nephritis and chronic endocarditis.

Physical Signs.—When the pericardium is coated with fibrin and there is little or no serum in the pericardial sac, there is usually a characteristic friction sound. This sound may be of a rubbing, grating, creaking, or whistling character, or may resemble an endocardial murmur. The sound may be loud or very faint. It is heard with the first or second sound of the heart, or with both, but is not always synchronous with these sounds. Rarely there is a presystolic friction sound. There is no fixed point of intensity of pericardial murmurs; they may be loudest at any point over the anterior surface of the heart; usually the sound is superficial.

Pericardial murmurs are to be distinguished from endocardial murmurs by their rubbing quality, their superficial character, their limitation to a small area, their changeableness in position and intensity from hour to hour, their greater intensity on pressure over the chest wall, and their want of synchronism with the heart sounds. They cannot always be distinguished from endocardial murmurs. They may be absent altogether.

When the pericardial sac is distended with fluid the distension may be symmetrical, or may bulge more in one direction than in another, so the shape of the area of precordial dulness varies. Fluid in the pericardial sac cannot always be distinguished from dilatation of the heart or from large productions of fibrin in the pericardial sac.

Rheumatic Pericarditis.—In different countries the frequency of pericarditis with rheumatism, and the comparative frequency of the cases with fibrin alone and of those with large exudations of serum, seems to vary. In New York pericarditis with fibrin alone is seen often enough, but the large exudations of serum are not very common.

Pericarditis occurs with both mild and severe rheumatic infections, and at any time in the course of the infection. The most obscure cases are those in which there is no inflammation of the joints and the pericarditis runs a subacute course.

In the cases with fibrin alone an increased frequency of the heart action, more or less precordial pain, and the friction sound are the only symptoms. With the distension of the pericardial sac with serum come the pressure symptoms and the dyspnœa.

CASE I.—Female, 27 years old, January 29.

Five days before admission to the hospital she had an attack of acute articular rheumatism. On the day of admission there was a systolic pericardial friction murmur, and on the next day a double friction murmur. The friction sound continued through the whole of her illness. She continued to get worse and died cyanotic and delirious.

Autopsy.—The pericardium is symmetrically dilated. The dilatation is due to a great deal of fibrin with but little serum. There are vegetations on the aortic and mitral valves. Both pleural cavities contain large quantities of clear serum. The lungs are compressed and œdematous. The abdominal viscera are congested.

CASE II.—Male, 16 years old, January 14.

He has had rheumatism in his feet and a slight cough for several months. Three weeks ago the cough became worse, with dyspnœa, cardiac palpitation, and pains in both sides of the chest. After he came into the hospital the dyspnœa was urgent, with marked cyanosis. The pulse was rapid, but the temperature was not over 99°. The urine contained no albumin. On the third day in the hospital he died.

Autopsy.—The anterior surface of the heart is entirely uncovered by the lungs. The pericardium extends up to the first rib and has the shape of a pericardial sac distended with fluid. The distension of the pericardial sac is due to the presence of a large quantity of fibrin with but little serum. The left ventricle is dilated, with thin walls. There is a fringe of small vegetations on the aortic and mitral valves. There are no minute changes in the walls of the ventricles. Both pleural cavities contain serum. In both lungs the bronchi are congested and contain muco-pus down to the small bronchi. In all the lobes there are a number of lobules of smooth hepatization. The liver and kidneys are congested.

Case III.—Female, 25 years old, December 30.

Five weeks ago her left knee and ankle became swollen and painful. Two weeks ago she suddenly developed sharp pain in the left mammary region, and any exertion caused severe dyspnœa. She also had fever, nausea, headache, prostration, sleeplessness, and orthopnœa.

Now, at the beginning of the third week of her endocarditis, she looks very badly, pale and cyanotic, with distressing dyspnœa, the heart's action rapid and feeble, the left foot swollen and painful, a loud, double friction sound over the entire precordial region, temperature 101°, pulse 120, breathing 38. Urine 1024, albumin present.

On the twenty-seventh day the friction sounds had disappeared; she could lie down, and felt much better.

On the thirty-first day she felt worse and there were subcrepitant râles over the lower lobe of the left lung.

On the thirty-fifth day she had continued to get worse, with dyspnœa, cyanosis, tongue brown and dry, marked prostration.

On the forty-fifth day she was in the typhoid state and there were dulness, bronchial breathing, and subcrepitant râles over the lower lobe of the right lung.

On the fifty-seventh day she was feeble and emaciated, but was beginning to improve.

On the seventy-seventh day there were flatness on percussion and bronchial breathing on the right side from the angle of the scapula down. Clear serum was drawn off with the needle.

On the eighty-third day she began to sit up.

On the one hundred and twelfth day she had a fresh attack of rheumatism involving both ankles and knees. This subsided in a few days. She was discharged entirely well on the one hundred and twenty-third day. The temperature was very irregular, but never over 102°.

Pericarditis with Lobar Pneumonia.—This does not seem to be an extension of the pneumonia, but an additional pneumococcus infection. In New York it is seen more frequently in some winters than in others. In most cases it gives few symptoms, but there are patients in whom the symptoms of the pericarditis are more marked than those of the pneumonia. Very few patients recover from the combination of lobar pneumonia and pericarditis.

Pericarditis with Pleurisy.—In these cases the inflammation seems to extend from the pleura to the pericardium. If the pleuare patches of pavement epithelium. The stroma is thrown into papillæ of irregular size and shape. The thickening of the walls of the bronchi is made up of connective tissue.

The walls of the medium-sized bronchi within the lungs are moderately thickened.

Through the lungs are patches of hepatization and quite a good deal of interstitial pneumonia.

There are firm white clots in the pulmonary arteries.

The heart, aorta, liver, stomach, intestines, spleen, kidneys, and uterus show no morbid changes.

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The lungs are emphysematous, the bronchi congested and coated with mucus.

The kidneys weigh together four ounces; they are nodular and cystic.

CASE VIII. *Pericarditis; Chronic Endocarditis.*—Male, 48 years old, December 19.

He has had scarlatina and rheumatism. For some time past he has had attacks of severe cough in paroxysms, sometimes with hæmoptyses. More recently he has suffered from frequent and severe headaches and occasional vomiting. For two weeks the cough and dyspnœa have been worse, and he has a good deal of pain over the liver.

Now pulse 92, temperature 97°, breathing 30; systolic murmur at the apex of the heart; severe pain over the liver; urine 1014, no albumin.

He continued with no fever, severe pain over the liver, and steady loss of flesh and strength, until he died one month later.

Autopsy.—The brain and its membranes are normal.

The pericardium is coated with fibrin; the mitral valve is stenosed and insufficient.

The lungs are congested and œdematous.

The liver and spleen are congested.

The kidneys, stomach, intestines, peritoneum, and vertebræ are normal.

Treatment.—For a rheumatic pericarditis the treatment of the rheumatism with salicylate of soda, etc., is a matter of much importance. With pneumonia and with double pleurisy the treatment of these conditions is necessary.

For the pericarditis, dry cups, blisters, or continuous cold over the precordial region, are of service. The feeble and disturbed heart action requires cardiac stimulants. If there is a considerable quantity of fluid in the pericardial sac we use cathartics or diuretics, or draw off the fluid with the aspirator. The point of election at which to insert the trocar is the fifth or fourth left interspace, about one inch from the sternum. In some cases the fifth right interspace at the sternal border is the best place. For my own part, I think that just inside the outer left limit of the line of dulness is the best place to tap.

This tapping of the pericardial sac must be done very carefully.

It is easy to wound the heart, and the withdrawal of the fluid may be followed by most alarming attacks of heart failure.

In the cases with purulent effusion aspiration may not be sufficient. Then it is necessary to resect a small portion of a rib, open the pericardial sac, and put in a drainage tube.

ENDOCARDITIS.

The endocardium is a connective-tissue membrane which lines the cavities of the heart and forms the larger part of the valves. It is a thin, dense, and elastic membrane, containing but few blood vessels. Its surface is covered by a single layer of endothelium. It becomes the seat of productive rather than of exudative inflammation. During fœtal life the endocardium of the right heart is inflamed: after birth usually the endocardium of the left heart. The valves are the portion of the endocardium most frequently affected, but that which lines the auricles and ventricles and covers the papillary muscles may be inflamed independently.

I do not think that it is possible at the present time to make any satisfactory classification of cases of endocarditis. Some years ago nearly all cases of endocarditis, whether acute or chronic, were considered to be of rheumatic origin. The acute cases formed part of the history of acute articular rheumatism: the chronic cases were considered to be "organic disease of the heart."

More recently has been brought into prominence the fact that in both acute and chronic endocarditis there are cases with little disturbance of circulation, but with marked infectious symptoms.

To this has been added the experience that different forms of bacteria are present in the larger number of cases. At first it seemed that the presence of bacteria accounted for the infectious symptoms. But as the same bacteria are found in the heart lesions, both with and without infectious symptoms, this explanation is not sufficient.

It seems to me the only plan just now is to try to describe endocarditis as [illegible]

Lesions.—In acute endocarditis there are three essential changes due to the inflammation:

1. A simple swelling of the endocardium. The basement substance is swollen and succulent, the cells are increased in size, the endothelium remains intact. Such a swelling of the valves makes them shortened and insufficient.

2. A production of small round cells in the thickness of the endocardium, with a falling off of the endothelium. If these cells become necrotic, ulcers are formed. Such changes in the valves cause insufficiency, narrowing, and roughening.

3. The formation of thrombi of different sizes, which adhere to the inflamed valves. These thrombi may obstruct the openings of the valves. It is a question how far the formation of these thrombi is due to the condition of the valves and how far to the unknown conditions which cause the formation of thrombi in veins.

The micro-organisms most frequently found are streptococci, staphylococci, and pneumococci, but a variety of others have been described.

There are two anatomical forms of chronic endocarditis:

1. The endocardium is thick and dense. There may be little, beaded prominences on its surface; it may be infiltrated with the salts of lime; but its surface remains smooth and thrombi are not formed on it. Such an endocarditis renders the valves rigid insufficient, or stenosed.

2. The endocardium is thickened; there is a growth of cells accompanied by necrosis, so that more or less of the endocardium is destroyed. Thrombi are formed, which adhere closely to the inflamed endocardium. There may be a deposition of the salts of lime in the inflamed endocardium and in the thrombi.

The same micro-organisms are found as are seen in acute endocarditis.

This description of the lesions applies to all the cases of endocarditis, although for convenience we describe different clinical groups. The groups are as follows:

Endocarditis with acute articular rheumatism.

Endocarditis with chorea.

Endocarditis with gonorrhœa.

Endocarditis with disturbances of circulation.

Endocarditis with infectious symptoms.

Endocarditis with both infectious symptoms and disturbances of circulation.

1. *Acute Endocarditis with Acute Articular Rheumatism.*

This is especially common in young persons. It occurs in both severe and mild cases. It usually follows the inflammation of the joints, but may precede it, or may occur without any joint lesion at all.

Although micro-organisms are found on the inflamed endocardium in rheumatic endocarditis, it seems probable that the inflammation is due to the poison of rheumatism, whatever that may be, rather than to the presence of the micro-organisms.

Symptoms.—The physical signs are regularly those of aortic obstruction and insufficiency and of mitral insufficiency, aortic systolic and diastolic and mitral systolic murmurs.

In many cases of rheumatism the endocarditis is of mild type. The only symptoms are the aortic or mitral murmur, some disturbance of the action of the heart, and a moderate rise of temperature.

But there are cases in which the endocarditis is mild but the rheumatic poison is severe. These patients are very ill with the rheumatism and are liable to have in addition pericarditis, pleurisy, and pneumonia.

In the more severe cases of endocarditis the disturbance of the heart's action is much more serious and regularly follows one of two types:

1. The heart's action is violent, tumultuous, and irregular; there is very marked venous congestion of different parts of the body.

The congestion of the pia mater produces delirium and stupor.

The congestion of the lungs gives labored breathing, and may go on to inflammation and consolidation of parts of the lungs.

The congestion of the pleura produces hydrothorax; that of the stomach, vomiting; that of the liver, enlargement of that organ; that of the peritoneum, ascites; that of the subcutaneous connective tissue, dropsy.

The congestion of the kidneys is attended with a diminished quantity of urine, which contains a little albumin and a few casts. If the urine is scanty for a long time the patients become anæmic and develop the symptoms of chronic uræmia.

2. The heart's action is feeble and rapid. There is not as much disposition to venous congestion, but the patients suffer from dyspnœa, they are very feeble, they are liable to attacks of syncope.

A very serious feature of rheumatic endocarditis is that the patients may have repeated attacks of rheumatism and with each attack there is a fresh endocarditis.

A large number of cases of acute rheumatic endocarditis recover, but very few without permanent changes in the valves.

In some the valves, although rendered insufficient, undergo no further changes, and the patients do well.

In some the valves, after remaining unchanged for years, finally become the seat of chronic endocarditis.

In some the acute endocarditis passes into the chronic form, and the patients grow worse with but little interruption.

Treatment.—The principal objects of treatment are the cure of the rheumatism and the relief of the bad heart action with the consequent disturbances of circulation. For the rheumatism the most efficient remedies are the salicylate of soda, the oil of wintergreen, the alkalies, antipyrin, phenacetin, and aspirin.

If the heart's action is exaggerated, tumultuous, and irregular, with excessive venous congestion, we employ venesection, wet cups or leeches, purging or sweating. Chloral hydrate, amyl nitrite, opium, and potassium iodide are often of much service.

If the heart's action is feeble and rapid we need the cardiac stimulants—digitalis, convallaria, caffeine, and alcohol.

2. *Endocarditis with Chorea.*

In a large number of cases of chorea there are systolic murmurs at the base or apex of the heart, sometimes associated with an increase in the size of the heart and with irregular heart action.

In some of these cases there is a true endocarditis, in others there is not. I do not know any certain way of determining which conditions exist, except by waiting for several months and seeing whether the evidences of valvular changes persist.

3. *Endocarditis with Gonorrhœa.*

There are cases of gonorrhœa in which the disease, instead of behaving like a localized inflammation, behaves like a general sepsis, with rises of temperature, prostration, cerebral symptoms, and inflammations of different parts of the body.

Of these secondary inflammations, those of the endocardium and of the kidneys are the most fatal.

CASE I. *Gonorrhœal Sepsis with Endocarditis.*—Male, 20 years old, August 4, 1900.

Six weeks ago he had a chill followed by fever; this has been repeated. He has had bad headaches, has been irrational, but has managed to keep about until yesterday.

Now, at the beginning of the seventh week, he is irrational; there is a purulent discharge from the urethra, which contains gonococci. There is a loud mitral systolic murmur, but the heart's action is regular and of fair force.

In the ninth week, hæmoglobin 80 per cent., red cells 4,106,000, white cells 11,400.

In the tenth week the right elbow became inflamed.

In the eleventh week, hæmoglobin 54 per cent., red cells 3,128,000, white cells 10,800.

During the whole time he was in the hospital the condition was that of acute sepsis with marked cerebral symptoms; temperatures 100° to 104°.

He died at the end of the eleventh week.

Autopsy.—The pia mater of the brain is a little thickened.

The lungs are œdematous.

There are soft vegetations on the mitral valve of the heart.

The spleen contains a white infarction.

The kidneys are large, with pale cortex, and a yellow infarction in one of them.

4. *Endocarditis with Disturbances of Circulation.*

These are the ordinary cases of chronic heart disease, in which all the symptoms seem to be due to the inability of the heart to carry on the circulation of the blood in a normal manner.

The inflammation involves most frequently the aortic and mitral valves, less often the tricuspid and pulmonary valves and the endocardium of the ventricles and auricles.

With chronic endocarditis are often associated myocarditis, arteritis, emphysema, chronic bronchitis, chronic nephritis, and contraction of the arteries.

Causes.—Chronic endocarditis may succeed acute endocarditis, either immediately or after a long interval. It may be caused by rheumatism, gout, or syphilis. It very often is a primary lesion. It is rarely due to the rupture of a valve by external violence.

The symptoms depend on:

The endocarditis.

The dilatation and hypertrophy.

The character of the heart's action.

The presence of thrombi on the endocardium.

The myocarditis.

The arteritis.

The contraction of the arteries.

The emphysema.

The chronic nephritis.

The general venous congestion.

1. *The Endocarditis.*—The inflammation may last for months or years, cease to be active, and no further changes be produced in the valves; or it may continue slowly and with intermissions up to the time of the patient's death, the valves becoming more and more damaged, and the inflammation extending to other valves; or the inflammation may be more active, the endocardium more rapidly and profoundly changed, with a febrile movement and loss of nutrition.

The changes in the endocardium, due to the inflammation, may produce cardiac murmurs.

Aortic stenosis gives a murmur with the first sound, and during the first silence. The murmur is loudest at mid-sternum opposite the third interspace. It is transmitted upward and to the right. It may be heard behind on the left side of the second, third, and fourth dorsal vertebræ. It may be loudest at the lower end of the sternum, or in the second interspace, on the right or left side. There may be well-marked aortic stenosis with no murmur.

Aortic insufficiency gives a murmur with the second sound, loudest at mid-sternum opposite the third interspace. It is transmitted down the sternum, and upward to the right. It may be loudest at the lower end of the sternum, in the left fourth interspace, or at the apex. It is a very constant murmur.

Mitral stenosis gives a presystolic or systolic murmur, or both. The presystolic murmur is heard just before the first sound. It is loudest at the apex and is usually circumscribed. The systolic murmur is loudest at the apex and is transmitted to the left. The murmurs of mitral stenosis are very inconstant, and often absent altogether.

Mitral insufficiency gives a systolic murmur, loudest at the apex, and transmitted to the left. This murmur is nearly always a constant one.

Ventricular murmurs are produced by roughening of the ventricular endocardium, or thrombi in the ventricles. They are systolic, loudest at the apex, and circumscribed. They are not constant.

2. *Dilatation and Hypertrophy of the Ventricles.*—Dilatation alone of the ventricles adds to the dangers of the endocarditis. Hypertrophy alone seems often to be of benefit. Hypertrophy and dilatation together, if not excessively developed, may also be of benefit. A heart which remains of normal size, or which becomes small when associated with advanced valvular changes, is apt to act badly.

Aortic stenosis is regularly followed by dilatation and hypertrophy, first of the left ventricle, then of the right.

Aortic insufficiency is followed by dilatation and hypertrophy of the left ventricle.

Mitral stenosis should be followed by dilatation and hypertrophy of the left auricle and the right ventricle. But there may be dilatation alone of either or of both ventricles; or dilatation and hypertrophy of the left ventricle; or the entire heart may be small.

Mitral insufficiency is followed by dilatation alone, or dilatation and hypertrophy of the left ventricle.

3. *The Character of the Heart's Action.*—Disturbances of the heart's action seem to depend upon the changes in the valves and walls of the heart; upon contraction of the arteries; and upon unknown causes.

The heart's action may be rapid, slow, feeble, forcible, intermittent, irregular, or tumultuous. The contractions of the heart may be so feeble that there are more apex beats than arterial pulsations.

Aortic and mitral stenosis, without contraction of the arteries, gives a feeble and irregular pulse; with contraction of the arteries, the pulse is tense, and the heart action often laboring. Aortic stenosis may give more ventricular contractions than heart beats.

Aortic insufficiency gives an exaggerated heart action, with dilated arteries, full of blood, but soft.

Abnormal sensations referred to the heart seem to depend upon its disturbed action.

There may be actual pain, referred to the heart and the left shoulder; the pain constant, produced by exertion, or paroxysmal Or there are feelings of constriction, or of displacement of the heart.

4. *Thrombi Attached to the Endocardium.*—These interfere with the heart's action. They may obstruct the orifices of the heart; fragments of them lodged in the cerebral arteries produce convulsions, paralysis, and aphasia.

5. *Myocarditis.*—Interstitial myocarditis, degeneration of the heart muscle, and disease of the coronary arteries are each one capable of rendering the heart's action feeble and irregular, of producing attacks of angina pectoris, and of causing sudden death.

6. *Chronic arteritis* affecting many of the arteries adds to the difficulties of the circulation. If it involves the cerebral

arteries the characteristic symptoms of cerebral arteritis may be developed.

7. *Pulmonary emphysema* is a frequent complication, and adds much to the dangers of the endocarditis.

8. *Venous congestion* takes a very large share in producing the symptoms of chronic endocarditis. It is due to the valvular lesions. the dilatation of the ventricles, the disturbed heart action. the contracted arteries. and the complicating emphysema or arteritis.

Congestion of the pia mater produces delirium. stupor. and convulsions.

Congestion of the lungs produces chronic bronchitis. hæmoptyses. the pneumonia of heart disease. œdema of the lungs. and hydrothorax.

Congestion of the stomach produces pain. gastric dyspepsia, vomiting. and vomiting of blood.

Congestion of the small intestines produces loss of flesh and strength. There is also gastric and intestinal flatulence.

The general congestion of the body gives subcutaneous dropsy and dropsy of the serous cavities.

The kidneys may be the seat of chronic congestion. of chronic degeneration. or of chronic nephritis.

With chronic congestion of the kidney the quantity of urine is diminished. its specific gravity is high. there is little or no albumin. the quantity of urea to the ounce of urine is sufficient. This change in the kidney seems to add but little to the symptoms.

The chronic congestion. if protracted. is succeeded by a chronic nephritis. The specific gravity of the urine falls. the quantity of urea is diminished. and albumin and casts make their appearance.

With chronic degeneration of the kidney the quantity of the urine is diminished. the specific gravity and the proportion of urea are unchanged. there is a moderate quantity of albumin. and a few casts. If the excretion of urine is scanty for a long time the patients may pass into the condition of chronic uræmia.

The chronic nephritis is diffuse and most frequently follows one of two types:

1. There is an abundant exudation of serum from the vessels of the kidneys. The urine is of low specific gravity and contains large quantities of albumin. The patients are anæmic, dropsical, and suffer from chronic uræmia.

2. There is little or no exudation from the blood vessels of the kidneys, but there is an extensive growth of interstitial connective tissue. The urine is of low specific gravity, contains little urea and little or no albumin. The patients are liable to attacks of acute uræmia with contracted arteries.

Cause of the Disease.—The disease may succeed one or more attacks of acute endocarditis, or it may be chronic from the outset.

In the chronic cases the symptoms are usually not developed until the disease has existed for some time. Then first one or two symptoms appear and others are added later.

The first symptom may be:

Dyspnœa on exertion.
Anæmia and loss of nutrition.
Cough and expectoration.
Cardiac palpitation and pain.
Stomach symptoms.
Dropsy.
Contraction of the arteries.

It sometimes happens that the patients go on for a long time with an endocarditis, but without symptoms. Then suddenly, with a pericarditis, a pleurisy, a bronchitis, or without discoverable cause, they develop a most distressing and urgent dyspnœa with contracted arteries. This dyspnœa continues and the patients die within a few days or weeks.

The endocarditis may stop at any time. It may go on slowly or quickly; it may be complicated by the diseases already mentioned.

The bad cases die with dyspnœa, or with dropsy, or with cerebral symptoms, or feeble and emaciated, or suddenly.

The prognosis is always to be made with a good deal of reserve. Persons in whom the cardiac symptoms are very marked, and whose condition seems almost hopeless, are capable of very great improvement under treatment. They may live for months or years longer than is expected.

In judging the prognosis we think of:

The extent and activity of the endocarditis.

The degree of the stenosis or insufficiency of the valves.

The condition of the ventricles.

The character of the heart's action.

The presence or absence of contraction of the arteries.

The character of the associated lesions.

If one only sees these cardiac cases during life, or only examines the lesions after death, the conditions seem comparatively simple. But if one both sees the cases and makes the autopsies for many years, one finds one's self confronted with very great difficulties in the exact appreciation of each case.

It is very difficult to tell how long an endocarditis has lasted before it begins to give symptoms. In many very old cases, as long as twenty years, there are hardly any symptoms and then the symptoms come on acutely.

When the symptoms do come on it requires great care to tell whether they are due to the narrowing of a stenosis, the dilatation of the ventricles, the failing compensation, the disturbed heart action, the contraction of the arteries, or the complicating nephritis.

It is very difficult to appreciate the character and extent of the valvular lesion, whether it is a simple thickening, or a cell growth with necrosis and thrombi.

The rule seems to be that cell growth and necrosis are more common in the aortic valves and simple thickening in the mitral valves.

Treatment.—It is impossible to lay down anything but the most general rules for treatment. The success depends on the experience and skill of the physician.

The principal indications are to check the progress of the endocarditis, to regulate the action of the heart and the contraction of the arteries, and to alleviate symptoms.

CASE II. *Endocarditis with Cell Growth and Necrosis of the Aortic Valves.*—Male, 23 years old, January 10, 1880.

He has never had rheumatism.

One year ago he had an attack of dyspnœa and vomiting which lasted for three days. After that he was well until five weeks ago. Then he became pale, feeble, had night sweats, pains in the chest and arms, constant severe headaches, and occasional vomiting.

On the third day of the attack the left foot and leg became swollen and painful.

One week ago he began to have cough and dyspnœa, became more feeble, and had one or two sharp chills every day.

Now he is feeble and emaciated, the skin dusky, the tongue coated and yellow, urgent dyspnœa, pulse 102 and hard, temperature 100°, breathing 28. There is an aortic diastolic murmur, loudest in the third interspace.

He grew steadily worse, with dyspnœa and expectoration of bloody, frothy mucus. The next day he died.

Autopsy.—The brain was not examined.

The right ventricle of the heart is a little dilated; the left ventricle is moderately dilated, with thin walls. There is a moderate cell growth between the muscular fibres. Two leaves of the aortic valve and part of the aorta beneath are nearly destroyed, and to the portions of the valves still left are attached large, soft, white vegetations extending up into the aorta.

Both pleural cavities are nearly full of serum. Both lungs show the changes of the pneumonia of heart disease with considerable red hepatization. The bronchi are full of bloody serum. The liver is small and firm.

The spleen is large and soft, and contains one yellow infarction.

The kidneys look normal to the naked eye.

CASE III. *Endocarditis with Cell Growth, Necrosis, and Calcification of the Aortic Valves.*—Male, 52 years old, March 22, 1882.

Three months ago he began to have cough with mucous sputum and dyspnœa.

One month ago he had to stop work.

For eight days he has been in bed with cough, dyspnœa, and sleeplessness.

Now he is emaciated, cyanotic; dyspnœa, cough, small hæmoptyses, heart's action feeble, double aortic murmur, temperature 97°.

Four days later he died suddenly.

Autopsy.—The brain was not examined.

The lungs are covered with old pleuritic adhesions; they are emphysematous; there is no congestion or œdema.

There are old pericardial adhesions. The aortic valves are ulcerated, partly calcified, and covered with vegetations. The ventricles are small; their walls are not thickened.

The liver is hard and congested.

The stomach and small intestines are congested.

The kidneys are atrophied and nodular.

Case IV. *Aortic Insufficiency with Large Vegetations Hanging Down into the Ventricle.*—Male, 30 years old, November 20.

When he was admitted he was not aware of the serious nature of his illness and did not know how long he had been sick.

He was pale and anæmic, with great dyspnœa, increased by exertion. The heart was much enlarged, with a diffuse, heaving pulsation. There was a loud diastolic murmur, most distinct at a point two inches to the left of the apex and transmitted to the back.

The dyspnœa continued; there was general dropsy; he became delirious, with a moderate rise of temperature, and died at the end of six weeks.

Autopsy.—The pericardium is covered with old adhesions. Both ventricles are dilated, without hypertrophy. The mitral valves are normal. The aortic valves are thickened and insufficient; to their edges adhere three large, polypoid vegetations which hang far down into the ventricle.

The lungs show the lesions of the pneumonia of heart disease.

The kidneys are in the condition of chronic congestion.

Case V. *Endocarditis with Cell Growth and Calcification of the Aortic Valves.*—Female, 20 years old, January 5, 1878.

Three months ago she caught cold and began to have cough with white sputum, sometimes streaked with blood, headache, vomiting, dyspnœa, palpitation, and pain over the heart.

Two weeks ago she went to bed, began to have dropsy of the feet, and the urine was nearly suppressed.

Now she is feeble and anæmic; the heart is dilated, with a loud double murmur all over it. There are subcrepitant râles over both lungs.

She gradually grew worse and died four days later.

Autopsy.—The brain was not examined.

Both ventricles of the heart are dilated, without hypertrophy. The aortic orifice is nearly closed by one leaf of the valve, which is large and calcified.

The lungs show the lesions of the pneumonia of heart disease and are œdematous.

The stomach and small intestines are congested and coated with mucus.

The liver is congested.

The kidneys are large, hard, pale, and nodular.

CASE VI. *Endocarditis with Cell Growth and Thrombi of Mitral Valve and Left Auricle.*—Male, 12 years old, February 9, 1884.

Five years ago he had rheumatism with cardiac complications, and since then has had several subacute attacks. For four months he has been much worse, with cardiac pain, dyspnœa, nausea, cough, and dropsy.

Now the heart is dilated, with a loud systolic murmur all over it, and irregular and tumultuous action. The urine contains albumin and granular casts.

He died comatose sixteen days later.

Autopsy.—The brain was not examined.

The pericardium is covered with old adhesions. The right auricle and both ventricles are dilated. The mitral valve is thickened and covered with vegetations. The endocardium of the left auricle is covered with vegetations.

Both pleural cavities contain serum. The lungs show the lesions of the pneumonia of heart disease.

The liver is large and congested.

The spleen is large and firm.

The kidneys are large, pale, and contain white infarctions.

CASE VII. *Endocarditis with Cell Growth and Necrosis of Aortic and Mitral Valves.*—Male, 23 years old, December 31, 1878.

Five years ago he had a chancre.

For two years he has had occasional palpitation of the heart.

For six weeks he has had pain and stiffness in the joints, cough, and palpitation of the heart.

For two weeks there has been dropsy of the legs and abdomen.

Now he is well nourished, good appetite, tongue clean, bowels regular, pulse 100, temperature 101°, breathing 24. Urine 1014, albumin, granular and epithelial casts.

There is a double aortic murmur with hypertrophy and dilatation of the left ventricle.

January 6 he had a chill, fever, and sweating.

One week later he had bloody expectoration.

At the end of two weeks he died.

Autopsy.—The brain was not examined.

The left ventricle of the heart is greatly hypertrophied and dilated; the right ventricle is a little dilated. The aortic valves are short and thick, with a long vegetation attached to one leaf. Another leaf is very much thickened and ulcerated, and behind is a cavity with roughened walls. The mitral valve with its tendons is thickened and ulcerated.

There is serum in both pleural cavities.

The lungs show the lesions of the pneumonia of heart disease.

The liver is large and nutmeg-colored.

The spleen is large and firm.

The stomach shows the changes of chronic gastritis.

The kidneys are large and white and show the changes of chronic nephritis.

CASE VIII. *Endocarditis with Thickening of the Mitral Valve Producing Insufficiency.*—Male, 25 years old, April 11, 1877.

In February he had pleurisy with effusion.

Two weeks ago he began to have dyspnœa and cramps in the abdomen.

Now he is cyanotic; vomits; has dyspnœa; the heart is dilated, with a systolic mitral murmur; the left pleural cavity is half-full of serum; there is general dropsy; temperature 98°-100°; urine 1022, trace of albumin.

He continued in about the same condition for six months, when he died.

Autopsy.—The dura and pia mater are congested.

Both ventricles of the heart are dilated, without hypertrophy; the mitral valve is thickened and insufficient.

The left pleural cavity is two-thirds full of serum and the lung is compressed. The right pleura is coated with connective tissue infiltrated with serum. The upper lobe of the right lung is in the condition of smooth red hepatization.

The peritoneum is congested and contains turbid serum.

The stomach and small intestine are congested and coated with mucus.

The liver is small and hard.

The spleen is large and firm.

The kidneys are large, hard, and smooth—chronic congestion.

CASE IX. *Endocarditis with Thickening and Calcification of Mitral Valve Producing Stenosis.*—Male, 45 years old, January 15, 1889.

May 29, 1888: He was in the hospital for a month with precordial pain, dyspnœa, dropsy of the legs, œdema of the lungs, rapid and tumultuous heart action, albumin in the urine.

After this he was much better until November 1. Then he came back to the hospital with the same symptoms, but after two weeks was much improved. He continued pretty well until January 15, 1889. Then he came back to the hospital with urgent dyspnœa, the heart dilated, irregular, and rapid, with mitral systolic murmur; temperature 99°; urine 1023, albumin, hyaline and epithelial casts.

The next day he died suddenly.

Autopsy.—The brain is normal.

The right ventricle of the heart is dilated and hypertrophied; the left ventricle is dilated, with thin walls. The aortic valves are a little thickened. The mitral valve is thickened, calcified, and stenosed.

The liver, spleen, stomach, and small intestine are congested.

The kidneys are large, hard, and show the lesions of chronic nephritis.

CASE X. *Endocarditis with Moderate Thickening of the Aortic and Mitral Valves; Cardiac Symptoms Out of Proportion to the Cardiac Lesions.*—Female, 39 years old, July 8, 1879.

No history of rheumatism or syphilis.

One year ago she had an attack of lobar pneumonia.

Three months ago she began to have dull pain in the left side, and cough with mucous sputum. The cough has grown worse and the sputum is sometimes streaked with blood.

Two months ago the urine became scanty; there was dropsy of the feet and legs, and dyspnœa on exertion.

One month ago she was confined. After her confinement she had dull precordial pain, dyspnœa, and palpitation.

Now, orthopnœa; pulse feeble and rapid; dropsy of both legs; urine 1033, albumin; an aortic regurgitant murmur.

August 6 she was discharged improved.

October 4, readmitted. For the past month she has had precordial pain, cough, several attacks of syncope, dropsy of the feet and legs.

Now there are dilatation and hypertrophy of the heart with a mitral systolic murmur.

October 7: Two attacks of syncope.

October 25: Vomits after meals; no dropsy; pulse feeble, but regular.

October 28: Pain in abdomen; bloody and watery diarrhœa.

December 5: All the contractions of the ventricles are not strong enough to be felt in the radial artery; 104 pulsations of the heart to 44 of the radial pulse.

December 30: She is steadily getting more feeble and is delirious.

January 19: She died suddenly.

Autopsy.—There is an increase of serum in the pia mater; no congestion.

The aortic and mitral valves are moderately thickened and insufficient. Both ventricles are dilated and hypertrophied. There are old pericardial adhesions. There are no minute changes in the walls of the ventricles.

The lungs are covered with old adhesions; there is a little old pigmented fibrous tissue at the apices.

The liver is congested.

The spleen is large and soft.

The kidneys show only degeneration of the epithelium.

CASE XI. *Chronic Inflammation of the Endocardium of the Left Ventricle.*—Female, 31 years old, May 25.

During the preceding winter she began to have cough with hæmoptyses and night sweats. She has had an eruption and ulcers of the skin.

For three months she has had nausea, vomiting, and dropsy of the feet.

Now there is a mitral systolic murmur; dropsy of the feet; urine contains a little albumin and hyaline casts.

June 4: There is flatness over the lower half of the right lung.

June 5 she suddenly became cold and pulseless.

June 8 she died.

Autopsy.—The brain is normal.

The heart weighs sixteen ounces. The endocardium of the left ventricle is thick, white, and in places roughened, with fibrin adhering to it. The mitral valve is insufficient.

Both pleural cavities are about half-full of serum.

Both lungs are partly aërated and œdematous. In the lower lobe of the left lung there is a small hemorrhagic infarction.

The liver weighs three pounds, nutmeg.

The kidneys weigh together eleven ounces; the Malpighian tufts are waxy.

CASE XII. *Chronic Endocarditis of the Left Auricle.*—Female, 34 years old, February 14, 1883.

For several years she has complained of palpitation, dyspnœa, and cough.

Last winter the cough was worse, with blood-stained sputum.

Seven weeks ago she had an attack of erysipelas of the face and neck.

Three weeks ago dropsy began in the legs and extended upward; the urine was scanty.

For two weeks she has been delirious at night.

Now there is general dropsy; great dyspnœa; delirium; pulse 72, small and feeble, temperature 100°, breathing 36; urine 1017, albumin and casts, fourteen to thirty-four ounces. The heart is feeble and irregular.

She improved, with less dropsy and less dyspnœa, until February 24, when the sputum became bloody and she died.

Autopsy.—The brain shows an increase of clear serum beneath the pia.

The right ventricle of the heart is dilated and hypertrophied; the left ventricle is normal. The aortic valves are a little thickened and insufficient.

The left auricle is very small; its entire wall is thickened; in some places it is half an inch thick, and this thickening extends to the mitral valve; the valve itself is but little changed. The whole condition is such as to be equivalent to a mitral stenosis.

The left lung is completely covered with old adhesions, and the pulmonary pleura is much thickened. The whole lung is small and leathery. The middle of the upper lobe is dense and hard, traversed by bands of connective tissue, and contains small, hard nodules.

The right lung is large and in the condition of pigment induration.

The liver is large, hard, and finely nodular.

The spleen is large and firm.

The stomach and small intestine are congested and coated with mucus. The kidneys are of medium size and show the lesions of chronic nephritis.

In the following table are given the principal symptoms and lesions of 263 fatal cases of chronic endocarditis.

ENDOCARDITIS.

TABLE OF 263 FATAL CASES OF CHRONIC ENDOCARDITIS WITH DISTURBANCES OF CIRCULATION. AUTOPSIES.

	Aortic stenosis.	Aortic insufficiency.	Mitral stenosis.	Mitral insufficiency.	Aortic stenosis and mitral insufficiency.	Aortic stenosis and mitral stenosis.	Mitral insufficiency tricuspid insufficiency.	Mitral stenosis pulmonary stenosis.	
Number	36	46	45	41	38	47	9	1	263
Both ventricles dilated	8	9	7	9	9	6	1	..	49
Both ventricles dilated and hypertrophied	3	7	9	4	9	23	3	1	59
Right ventricle dilated	..	..	5	..	..	2	3	..	10
Right ventricle hypertrophied	1	..	8	1	1	1	1	..	13
Left ventricle dilated	2	10	3	5	3	2	..	..	25
Left ventricle hypertrophied	9	12	5	15	10	6	..	..	57
Pneumonia of heart disease	9	16	28	16	13	31	5	1	119
Œdema of the lungs	8	18	9	12	12	9	5	..	73
Emphysema	6	8	5	7	7	3	..	..	36
Bronchitis	6	7	11	6	3	7	1	..	41
Chronic nephritis	21	22	17	27	22	23	..	1	133
Chronic congestion of the kidney	9	18	20	9	11	17	8	..	92
Normal kidneys	6	2	6	3	5	6	..	..	28
Cerebral apoplexy	1	1	2	2	1	2	..	..	9
Cerebral embolism	3	..	2	2		1	..	..	8
Dropsy	17	33	35	25	26	31	5	1	173
Delirium	4	7	4	2	4	8	1	..	30
Convulsions	..	1	1	2	1	4	..	..	9
Coma	5	6	6	7	4	3		..	31
Dyspnœa	18	35	31	23	28	34	6	..	175
Hæmoptysis	4	8	8	4	5	10	..	..	39
Cough	7	18	26	12	15	21	2	..	101
Sudden death	1	10	4	6	5	9	..	1	36

5. *Endocarditis with Infectious Symptoms.*

Many of these cases have been observed and described, but their real nature is still obscure.

A person who has had rheumatic endocarditis, chronic endocarditis, lobar pneumonia, a focus of septic infection, or has apparently been in good health, may at some time develop symptoms of acute, subacute, or chronic sepsis with a cardiac murmur.

Disturbances of the heart action or of the circulation are not present unless they are due to the pre-existing chronic endocarditis.

Lesions.—Either the mitral, the aortic, or the tricuspid valve, the endocardium of the left auricle or of the right ventricle, may be inflamed. The inflamed endocardium is covered with a thin or thick layer of soft material composed of fibrin and micrococci, usually the ordinary streptococci and staphylococci of suppuration or the diplococcus pneumoniæ. The endocardium beneath is swollen, infiltrated with cells, often ulcerated. If the malignant endocarditis affects a heart already the seat of chronic endocarditis, the old lesions persist.

Portions of the soft matter on the inflamed endocardium often become detached and are lodged as emboli in the arteries of the brain, lungs, heart, spleen, and kidneys. These emboli are infectious and set up an inflammation of the surrounding tissues. The liver, spleen, and kidneys are swollen and their cells degenerated.

As in other septic conditions, there are often fibrinous and purulent inflammations in different parts of the body, and thrombi in the veins.

It is still an open question whether in these cases the endocarditis causes the sepsis, or whether this inflammation is itself only one of the evidences of a general poisoning.

The cases seem to group themselves into:

1. *Subacute cases which terminate in death or recovery.*

In these patients the symptoms are: a gradual loss of flesh and strength; a diminution in the quantity of hæmoglobin and the number of red blood cells; an increase in the number of white blood cells; an endocardial murmur and more or less afternoon fever. The symptoms continue for weeks or months and terminate in death or recovery.

CASE XIII. *Endocarditis with Infectious Symptoms, Terminating in Recovery.*—Male, 35 years old, May 27, 1903.

Usually a strong, robust man.

For several months he has not felt well and has had queer feelings in his arms, but has continued to be actively engaged in a large business. He sleeps well, has no gastro-intestinal or pulmonary symptoms, has noticed no fever.

Hæmoglobin 75 per cent., white cells increased in number. The heart is not enlarged; its rhythm is good. There is a loud mitral systolic murmur.

June 2: The systolic murmur is not as loud.

June 17: The murmur is now loudest in the second left interspace. He has gained seven pounds in weight and feels better. Hæmoglobin 90 per cent., white cells 10,000.

June 29: The murmur is louder; he has a temperature of 100° in the afternoon; hæmoglobin 78 per cent.

July 17: The murmur has disappeared; no more fever; hæmoglobin 72 per cent.

September 17: There is no murmur; he feels perfectly well; white cells 9,000, hæmoglobin 75 per cent.

CASE XIV. *Endocarditis with Infectious Symptoms; Subacute Case, about Thirty Days' Duration of Symptoms.*—Male, 45 years old, an orderly in the hospital.

September 6, 1883, he was admitted to the ward with an irregular febrile movement, 96°-102°, and the story that he had been gradually getting feeble and anæmic. He developed a systolic murmur at the base of the heart, continued to have an irregular temperature, gradually became more feeble, and died on October 7.

Autopsy.—The brain was not examined.

The body is emaciated.

There is a large, soft vegetation of one leaf of the aortic valve, with ulceration of the valve and of the soft parts beneath. There are colonies of micrococci in the vegetation.

The bronchi contain pus; there is a little fibrin on the left pulmonary pleura.

The liver is large; commencing cirrhosis.

The spleen is large and contains red and white infarctions.

The kidneys are of medium size; chronic nephritis.

CASE XV. *Endocarditis with Infectious Symptoms, running a Subacute Course of about Five Weeks.*—Female, November 12, 1884.

For three weeks she has suffered from headache, pain in the back, nausea and vomiting.

Now temperature 101°, pulse 90; tongue coated; tonsils and pharynx swollen; slight redness, swelling, and tenderness of the left ankle. There is a loud systolic murmur over the whole precordial region.

November 15: Temperature 102°-104°, pulse 130 and feeble, breathing 30-46. The skin is cyanotic; the tongue is dry and coated; there are moist râles over the lower lobe of the right lung.

November 18: Temperature 99°-101°, pulse 100. She looks as if she were suffering from an infectious disease; pains all over the body; will not eat or speak.

November 26: She lies quietly in bed; takes no notice of anything; the urine and fæces are passed involuntarily; temperature 100°-103°.

November 30 she died.

Autopsy.—There is no pericarditis. On the aortic valves there are a few small vegetations. The mitral valves are a little thickened, with small ulcers and small adherent thrombi.

The lungs are congested, with small patches of red hepatization in the lower lobes.

The liver is fatty.

The spleen is large and soft.

The kidneys look normal to the naked eye.

CASE XVI. *Endocarditis of the Left Auricle with Infectious Symptoms, running a Subacute Course of about Nine Weeks.*—Female, 17 years old, September 16, 1884.

Six weeks ago she began to have severe headache and pains all over the body.

Now she looks stupid; complains of constant headache; is very anæmic; there is a mitral systolic murmur.

She continued to grow steadily weaker and more emaciated, with irregular temperatures from 98° to 103°, until she died three weeks later.

Autopsy.—The brain is somewhat œdematous.

The mitral valve is partly calcified, with a small thrombus adherent to it. On the endocardium of the left auricle is a patch, one inch in diameter, studded with small thrombi. There are colonies of micrococci in the thrombi.

The lungs, liver, and uterus are normal.

The spleen is large and contains a white infarction.

The kidneys look normal.

2. *Acute septic cases.*

These run a short course with the symptoms of an acute and virulent infection. Repeated chills, an irregular febrile movement, delirium or stupor, cough with bloody sputum, a rapid and feeble heart, ecchymoses in the skin, increased leucocytosis, a dry tongue, and rapid emaciation are the principal features.

The disease is apt to prove fatal inside of two weeks.

Case XVII. *A Thrombus of the Mitral Valve as Part of a General Postpartum Sepsis.*—Female, 24 years old, July 14, 1876.

Two weeks ago she had a normal confinement. She got up on the third day, had some fever, and went to bed again. Since then she has been up and about.

Now pulse 120, temperature 105°; the uterus as large as a fœtal head.

July 27: Retention of urine, diarrhœa, and a good deal of pain in both legs.

July 28: She vomited blood; there was a loud mitral systolic murmur; the legs were cold; there was no pulsation in the femoral arteries.

The temperature ran from 100° to 105°, with chills nearly every day.

August 1 she died.

Autopsy.—The brain is normal.

The right ventricle of the heart is dilated. There is a large thrombus attached to the mitral valve, otherwise the valves are normal.

The spleen weighs twelve ounces and contains several infarctions.

The kidneys show the changes of acute degeneration.

The stomach and intestines are normal. The uterus is large; its inner surface is coated with pus; a portion of decomposing placenta is adherent to the fundus.

The aorta at its bifurcation is completely filled with a firm white thrombus, which extends into both common iliac arteries.

Case XVIII. *Old Endocarditis, Recent Endocarditis with Infectious Symptoms of Nine Days' Duration.*—Male, 43 years old, October 13, 1878.

The man was well and at work until five days ago, when he complained of general malaise and vertigo.

Four days ago he had chills and vomiting.

Now he is well nourished; tongue brown and dry; delirious; small ecchymoses in the skin; moist râles over both lungs; pulse 116-132, temperature 102° to 103°, breathing 34-44. He continued in the same condition, became comatose, and died four days later.

Autopsy.—Beneath the pia mater of the convexity there is a thin layer of fluid blood. There are groups of round and polygonal cells and a few threads of fibrin in the pia.

There are ecchymoses in the pericardium. The aortic valves are thick, calcified, stenosed; their ventricular surfaces are covered with vegetations, and beneath these the valves are ulcerated.

There are ecchymoses in the pleura; the bronchi are congested and coated with muco-pus, the lungs are congested. The liver weighs five pounds and is cirrhotic.

The spleen is large and contains soft recent infarctions.

The kidneys are large, contain old and recent infarctions and small foci of pus.

The spinal cord and its membranes are normal.

CASE XIX. *Endocarditis with Infectious Symptoms and Acute Nephritis of about Twenty-one Days' Duration.*—Female, 18 years old, January 24, 1883.

She came into the hospital with acute tonsillitis; urine 1023, a great deal of albumin, with blood and epithelial casts. She seemed to be very little sick, gave no other symptoms until February 8, when she had a chill followed by fever, sweating, nausea, pain in the back, drowsiness, cough with profuse brownish and bloody sputum.

The legs are dropsical; urine 1014, much albumin, blood and many casts. On March 3 there was an eruption of red blotches all over the body, which lasted for five days. The dropsy disappeared.

The temperature ran from 100° to 104°, pulse 96-168. She passed into the typhoid state and died March 13.

Autopsy—The brain was not examined.

On the surfaces of the aortic and mitral valves are small soft vegetations; otherwise, to the naked eye, the valves look normal. The endothelial cells covering the valves are increased in number, and just beneath the vegetations there is a cell growth and splitting-up of the basement substance.

The right pleura is nearly full of turbid serum; the pleura is coated with fibrin and pus.

The peritoneum is coated with fibrin and contains a little brownish serum.

The liver is normal.

All of the spleen, except a little of the upper end, is necrotic and softened; all its arteries are full of firm, white thrombi.

The stomach, intestines, uterus, aorta, and large veins are normal.

The kidneys show the lesions of acute productive nephritis.

CASE XX. *Endocarditis with Infectious Symptoms, Occurring in the Course of a Chronic Endocarditis, the Symptoms lasting for Ten Days.*—Male, 60 years old, after not feeling well for several days, on April 24, 1884, in the afternoon, had a severe chill followed by a temperature of 106°.

After this he had chills at irregular intervals, the temperature running between 99° and 106°.

On April 30 I saw him for the first time. The face was drawn, the tongue coated; he was apathetic, but could recognize persons; there was a loud double murmur over the whole heart; the heart's action and pulse were fairly good.

The next day he died.

Autopsy.—The body is emaciated. The brain was not examined.

The aortic valves are thick and hard; one leaf is very much thickened and covered with vegetations. The mitral valve is thick, hard, and insufficient. The wall of the right ventricle is thickened: the left ventricle is dilated and hypertrophied.

The lungs are congested and very œdematous.

The liver is fatty.

The kidneys are of medium size, nodular, with atrophied cortex.

3. *Cases lasting for a number of months, with afternoon fever as the principal symptom.*

In these cases the most noticeable symptom is the fever. Every day there is an afternoon temperature of from 100° to 104°, while the morning temperature is from 97° to 99°. With the fever there may be chilliness and sweating. There may be paroxysms, like those of ague, with chill, fever, and complete intermissions. A secondary anæmia and a moderate leucocytosis are usually present.

The patients are at first not confined to bed, but gradually lose flesh and strength. There is a cardiac murmur, but no other cardiac symptoms, unless a chronic endocarditis already exists. There are often days and weeks of considerable improvement and less fever. But then the fever comes on again and the patients get worse. They continue in this way for from six to twenty-four months and finally die feeble and emaciated.

CASE XXI. *Endocarditis with Infectious Symptoms: Afternoon Fever for Eight Months.*—Male, 50 years old, September 6, 1903.

Seven and a half months ago he began to feel sick and tired, to lose flesh, strength, and appetite.

Seven months ago he went to bed with fever, 98°-101° in the morning, 101°-102½° in the afternoon. He was kept in bed, on a fluid diet, for three months with the diagnosis of typhoid fever. Then he got up and went back to his business, at which he has continued ever since. He does not feel very badly, but always has an afternoon fever of from 101° to 104°.

He now weighs 150 pounds; before he was sick he weighed 200.

The lungs are normal. The left ventricle of the heart is hypertrophied and dilated; there is a double aortic and mitral systolic murmur. The radial arteries are thickened. The liver is not enlarged; the spleen is moderately enlarged.

Blood—hæmoglobin 30 per cent., red cells 3,176,000, white cells 10,000.

September 18 he reported that the afternoon fever was the same. He had gained a little in weight, but there was some dropsy of the legs.

4. *There are patients in whom the endocarditis produces both disturbances of circulation and evidences of infection.*

CASE XXII. *Endocarditis with Infectious Symptoms and Disturbances of Circulation.*—Female, 23 years old, March 28, 1900. No history of rheumatism or syphilis.

Six weeks ago she developed cellulitis of the right hand, which later extended up to the elbow and then subsided, leaving only some stiffness.

Three weeks ago she began to have irregular chills and fever, with some disturbance of the heart's action.

Five days ago she began to have dropsy of the feet.

Now, on the twenty-second day, there is a loud systolic murmur at the apex, transmitted to some distance. Both feet are œdematous, with hemorrhagic spots on one. Urine 1022, with a trace of albumin. Temperature 102°, pulse 120, breathing 32.

On the twenty-third day the œdema of the feet had disappeared. Hæmoglobin 42 per cent., red cells 2,862,000, white cells 17,200.

On the twenty-eighth day she was getting more septic and at the same time had more dyspnœa and more venous congestion. The morning temperatures ran at about 100°; the evening temperatures at 103°, with repeated chills.

On the thirty-first day she died.

Autopsy.—The pericardium contains clear serum. Both ventricles of the heart are dilated. The aortic valves are extensively ulcerated and covered with large vegetations.

Both pleural cavities are distended with clear serum; the lungs show the lesions of broncho-pneumonia.

The spleen contains red and white infarctions.

The liver is congested.

The kidneys look normal to the naked eye.

THROMBI IN THE HEART CAVITIES.

There is hardly any subject more obscure than that of thrombosis; all that is known on the subject is stated by Professor Welch in his article on "Thrombosis" in "Allbutt's System of Medicine."

It is very common to find, after death, in the heart cavities yellow, succulent clots. They are most common and of firmest texture in persons who die of acute inflammatory diseases. They may adhere quite firmly to the walls of the heart and extend in long cords into the vessels. They are formed in the last hours of life and just after death. They are of no clinical or pathological importance.

True thrombi are formed in the heart during life.

Some of them are adherent to the diseased endocardium and form part of the lesions of acute endocarditis.

Others are formed in the heart cavities without much relationship to endocarditis. They are in the form of flattened masses firmly adherent to the endocardium; or of rounded bodies in the spaces between the trabeculæ; or have a polypoid shape, attached by a narrow pedicle; or are free in the heart cavities.

They are firm, dry, and of a whitish color. They may soften and break down at their centres so as to look like cysts filled with pus, they may become calcified, or they may be changed into organized connective tissue.

Clinically they present themselves in two ways:

1. They are formed within a few weeks before death, are accompanied with great cardiac dyspnœa, and are a cause of death.

2. They are formed a long while before death, give very irregular cardiac symptoms, and are usually recognized only at the autopsy.

CASE I. *Thrombosis of the Heart after Typhoid Fever.*—Male, 42 years old, a lawyer. He has always been strong and well, and has had no cardiac symptoms, except that for a long time he has had a systolic murmur at the base of the heart.

During February, 1903, he went through a mild four-weeks typhoid fever and made a good convalescence.

The second week in March he went to Lakewood.

On March 17, in New York, on his way home, he had an attack of cardiac discomfort and dyspnœa.

On March 18 he went home. His physician then found some enlargement of the heart, pulse 80, cardiac dyspnœa. He was given bromides and digitalis and allowed to walk in the street.

March 29 the dyspnœa was decidedly worse.

March 31 I saw him. He was then unable to lie down. As he sat in his chair there was no appearance of dyspnœa. He had no cerebral or gastric symptoms. The legs and feet were œdematous. The breathing was easy and regular; there were subcrepitant râles over the lower lobes of both lungs.

The heart was much enlarged towards the left side, more dilatation than hypertrophy; its action was too forcible, regular, 100 to the minute. There was a loud systolic murmur over the whole heart, extending into the large vessels. The radial pulse was 100, of fair force, of digitalis quality.

I made the diagnosis of thrombosis of the left ventricle of the heart.

He died the next day.

There was no autopsy.

Case II. *Thrombosis of the Heart with Chronic Miliary Tuberculosis of the Lung.*—Male, 40 years old, came into the hospital with the symptoms of chronic phthisis. He was emaciated, but at first did not seem very sick. Then he began to vomit and vomited constantly for a week.

On September 16 I found him lying flat in bed, cyanotic, with no radial pulse, but with pulsation in the carotid and femoral arteries. The arms were cold; the legs were warm; there was no perspiration. He was perfectly rational and complained of nothing but want of breath. The heart was acting fairly well; the heart sounds were distinct; there was no murmur. I made the diagnosis of cardiac thrombosis. He remained in the same condition for several hours and then died.

Autopsy.—The brain was not examined.

The left lung is thickly studded with old miliary tubercles.

The heart is of medium size; the walls of the ventricles are normal; the mitral valve is a little thickened. The right cavities are completely filled with a yellow, smooth clot in which are embedded small, white, granular thrombi. Similar mixed clots extend into the pulmonary artery and vein. The left ventricle is contracted and nearly empty. A yellow and red clot, with white thrombi embedded in it, begins in the ventricle just below the aortic valves and extends into the arch of the aorta. The subclavian and carotid arteries are empty.

Case III. *Thrombus of the Left Ventricle; Embolus of the Left Middle Cerebral Artery.*—Male, 45 years old, April 6, 1875.

No history of rheumatism. He had always been well until the fall of 1873. Then, while sitting on his bench, he suddenly became cold and paralyzed on the right side. He did not fall or lose consciousness; his speech was somewhat affected. By the end of three months he recovered entirely, but the right arm remained weak.

March 28, 1875, he lay down in the evening. At 10 p.m. he got up and fell on the floor, but was able to get up again and undress himself.

March 29, in the morning, he walked out of the house. At 11 a.m. he fell on the floor, totally paralyzed on the right side and aphasic, but did not lose consciousness.

April 6 he was admitted to the hospital. There was right facial paralysis, complete loss of motion and sensation on the right side, and complete aphasia. There was no heart murmur.

April 22: There was thrombosis of the veins of the right arm and leg. There was a mitral systolic murmur.

July 7: The right heel was becoming gangrenous.

July 25: The temperature up to 104°, with dyspnœa and coarse râles over both lungs.

August 4 he died.

Autopsy.—There is a firm, old plug of fibrin in the two large branches of the left middle cerebral artery. The outer portion of the left corpus striatum, optic thalamus, and adjoining brain tissue, including the second and third frontal convolutions, are softened.

The heart is covered with old pericardial adhesions. The wall of the left ventricle is thin and soft. There are no changes in the valves. There is a large, firm, old thrombus free in the cavity of the left ventricle.

There is an infarction in the spleen. In the lower part of the right lung and the upper part of the left lung there are spots of gangrene and cheesy hepatization.

Case IV. *Thrombi in the Left Ventricle and Right Auricle, with Rheumatism.*—Female, 29 years old, June 16, 1870.

For seven years she has had pains in the joints of the fingers.

For two years she has had dyspnœa on exertion.

June 10 she had a chill and one instep and one knee became red, tender, and painful. Now the joints named are inflamed.

August 5 she developed severe dyspnœa and died suddenly.

Autopsy.—The brain was not examined.

There are large, soft vegetations attached to the mitral valve.

There is a firm, white, dry thrombus in the left ventricle, and another in the right auricle.

The lungs are in the condition of pigment induration and are œdematous.

The spleen contains a large infarction.

The liver and kidneys are normal.

CASE V. *Thrombi in the Right Ventricle with Chronic Pulmonary Phthisis.*—Female, 24 years old, November 19, 1869.

She has had the ordinary symptoms of chronic pulmonary phthisis for two years.

February 21, 1870, she died.

Autopsy.—The lungs show the ordinary lesions of chronic miliary tuberculosis with large bronchiectasiæ.

Both ventricles of the heart are dilated and hypertrophied. The right ventricle contains two thrombi. One is of rounded shape, the size of a filbert, with a soft, puriform centre. The other is flat, laminated, and adherent to the wall of the ventricle near the apex. There is also a long vegetation attached to the tricuspid valve.

The liver is fatty.

The Malpighian tufts of the kidneys are waxy.

CASE VI. *Thrombus of the Left Auricle.*—Male, 50 years old, April 21.

It is said that he has had rheumatism for four weeks.

Now there is no evidence of rheumatism; temperature 104°, pulse 100.

April 23 he had a chill; temperature 97° to 103°, pulse 66-100.

April 24 he got out of bed in the night and wandered about the ward; temperature 98°-97°, pulse 66-70. There is a systolic murmur at the apex.

May 13 he died.

Autopsy.—The pia mater is opaque, with an increase of serum. The cerebral arteries are normal. On the surface of the left hemisphere, at the outer and posterior part of the middle lobe, there is a spot about one inch in diameter, soft and yellow.

The heart weighs fourteen ounces; the right cavities are normal. The wall of the left auricle is thickened. A thrombus, about one inch long, is attached to the outer edge of the mitral orifice and the auricular surface of the mitral valve. Its base is covered with a smooth membrane continuous with the endocardium; the rest of it is ragged. The left ventricle is a little dilated.

The lungs show only the lesions of bronchitis.

The liver weighs five pounds ten ounces.

The spleen is large and contains two large infarctions.

The kidneys weigh together eleven ounces and contain small infarctions.

CASE VII. *Thrombus of the Left Auricle.*—Female, 32 years old, April 7.

She has been intemperate for years. One week ago she had chill, fever, dyspnœa, and pain in the left side.

April 11 two pints of bloody serum were aspirated from the left chest.

April 13 she died.

Autopsy.—The brain was not examined.

The heart weighs nine ounces. In the left auricle, firmly attached to the mitral valve and nearly closing it, is a rounded, firm, white thrombus.

The left pleural cavity is filled with purulent serum. The lung is compressed and coated with fibrin. In both lungs are hemorrhagic infarctions surrounded by zones of pneumonia.

The liver is congested.

The kidneys weigh together twelve ounces; chronic nephritis.

CASE VIII. *Organized Thrombi of the Right Auricle.*—The patient was a middle-aged woman without history.

Autopsy.—The right auricle and ventricle are both dilated. Attached to the inner surface of the right auricle, at a point half an inch from the tricuspid valve, and close to each other, are two thrombi. One of them is nearly spherical, two and a half inches in diameter, has a small round pedicle firmly attached to the wall of the auricle, and is entirely contained in the cavity of the auricle. The other thrombus is of ovoid shape, projects through the opening of the tricuspid valve into the ventricle, is partly calcified, and its free end is broken and ragged.

The thrombi present a yellow, semi-translucent appearance and are of firm, elastic consistence. Blood vessels containing blood can be seen in them with the naked eye. On the outer surface of the thrombi is a thin membrane continuous with the endocardium.

The thrombi are composed of blood vessels in considerable numbers, arteries and capillaries containing blood; a basement substance partly fibrillated, partly gelatinous, containing round, stellate, and fusiform cells, and in places calcified.

HYPERTROPHY AND DILATATION OF THE HEART WITHOUT ENDOCARDITIS.

1. From excessive and prolonged muscular exertion.

The left ventricle alone, or both ventricles, are hypertrophied or dilated, or both dilated and hypertrophied.

To produce these changes in the heart the muscular exertion must be excessive and prolonged, so we find them most frequently in soldiers, athletes, and laborers.

In the milder cases there is only hypertrophy of the left ventricle. The patient has the feelings of pain, constriction, or displacement of the heart, with rapid heart action.

These patients usually do well. It is only necessary to make them lead a regular life and sometimes to take digitalis.

In the more severe cases there is dilatation alone, or dilatation with hypertrophy. There are pains over the heart, rapid and feeble heart action, dyspnœa on exertion, and loss of flesh and strength. These cases are always serious, and not all of them recover.

It is necessary to keep the patients in bed and to use cardiac stimulants.

2. From disease of the lungs.

Emphysema, chronic phthisis, interstitial pneumonia, pleurisy, and deformities of the chest may produce dilatation, first of the right ventricle, and afterward of the left.

3. Exophthalmic goitre, or prolonged palpitation of the heart, may be followed by hypertrophy of the left ventricle.

4. Acute or chronic nephritis, chronic inflammation of the aortic system of arteries, and congenital narrowing of the aorta may produce hypertrophy of the left ventricle.

5. Simple anæmia may be attended with temporary dilatation and hypertrophy of the left ventricle, or of the right auricle and ventricle.

6. There is an important group of cases of dilatation of the ventricles without hypertrophy, of which the causation is very obscure.

Usually both ventricles are dilated, but sometimes only the left. The valves are normal, but their orifices may be so large that they are insufficient. The walls of the ventricles are normal, or the muscular fibres are degenerated, or there is a growth of cells between the fibres. Thrombi may be formed in the cavities of the ventricles. Venous congestion of the viscera is soon established, dropsy somewhat later.

Of the causes of this heart lesion we are ignorant. It is often enough developed in young and healthy adults. In some cases there is a distinct history of sudden muscular exertion; in others no exciting cause can be discovered.

In the more acute cases the invasion of the symptoms is sudden.

The patients complain of pain in the epigastrium, they become unconscious, and they vomit; or the first symptom is urgent cardiac dyspnœa. After this the heart's action is rapid, irregular, and feeble. The physical signs are those of dilatation of the heart. There is constant and distressing dyspnœa; there may be cough and hæmoptyses; the patients lose flesh and strength, and die at the end of a few weeks.

In the more chronic cases the invasion is less sudden and decided. The patients suffer from rapid, feeble, and irregular heart action, dyspnœa, cough and hæmoptyses, marked dropsy, and loss of flesh and strength. They may live for several months.

The treatment of these cases is eminently unsatisfactory. We are unable to improve the action of the heart, and the best that we can do is to palliate the patient's suffering with opium.

CASE I. *Dilatation of the Heart with Gastric Symptoms of about One Year's Duration.*—Male, 26 years old, laborer, October, 1874.

Was perfectly well until one year ago. Then he had an attack of epigastric oppression, followed by loss of consciousness and convulsive movements of the extremities lasting for several minutes. After half an hour he began to vomit and continued to vomit for several days.

Since then he has had four similar attacks at intervals of from two weeks to six months. No cough, dyspnœa, palpitation, or œdema. He has been treated for epilepsy.

Now he is a large, muscular, robust man, of florid color; good appetite; pulse and temperature normal; urine 1030, no albumin.

October 6, at 10 P.M., he awoke from his sleep with the feeling of a weight resting on his stomach; was much agitated; pulse 190; no dyspnœa.

October 7 he began to vomit a transparent, watery, greenish fluid in large quantities. He lies flat in bed, without dyspnœa, very restless, with exaggerated heart action and severe pain in the epigastrium.

October 9 he stopped vomiting.

October 11 he was up and about, but weak and pale.

October 15 he fell to the floor unconscious, but recovered in a few minutes. He soon began to have pain in the epigastrium and to vomit. The pain and vomiting continued until October 17.

October 19 another short attack of unconsciousness followed by pain and vomiting.

October 21: The vomiting continues; pulse 48; the heart's impulse is irregular and diffuse; the area of precordial dulness is enlarged.

After this he improved until November 5. Then the radial pulse was 30 and the heart beat 154; no murmur. After this he had epigastric pain and vomiting, the heart very rapid and feeble, but no dyspnœa.

November 15 he died.

Autopsy.—The brain is normal.

The heart weighs two pounds. The aortic, mitral, and tricuspid valves look normal, but are insufficient to water. Both ventricles are very much dilated.

The mucous membrane of the stomach is intensely congested.

The lungs are congested and œdematous.

The liver and spleen are normal.

The kidneys weigh together twenty-two ounces; they are not hard or congested.

CASE II. *Acute Dilatation of the Heart of about Twenty-five Days' Duration.*—Male, 26 years old, gardener, October 23, 1872.

With the exception of chills and fever, he has not been sick until the present attack. His habits are good; he has not had syphilis or rheumatism.

Two weeks ago, while lifting a wheelbarrow, he was suddenly seized with dyspnœa, so that he dropped the barrow. After this the dyspnœa continued, so that he could hardly lie down at all, but there was no pain.

Now there is marked dyspnœa, cough, irregular pulse, normal temperature. The heart is dilated; its action is feeble and irregular; there are no murmurs. There are dulness and diminished breathing over the lower lobes of both lungs.

He continued to have urgent dyspnœa, with irregular and feeble heart action, until November 3, when he died.

Autopsy.—There is a moderate increase of serum in the pia mater of the brain.

Both pleural cavities are half-full of clear serum. Both lungs are dry and leathery, with muco-pus in the bronchi.

The pericardial sac is half-full of clear serum.

The heart is of globular shape. The right auricle is dilated and full of blood. Both ventricles are dilated and contain red clots. The wall of the left ventricle is five-eighths of an inch thick; the wall of the right ventricle is one-eighth of an inch thick. I could find no minute changes in the walls of the ventricles. There are no changes in the valves.

The liver is large and congested.

The spleen is large and congested.

The stomach and kidneys are congested.

CASE III. *Chronic Dilatation of the Heart with Dropsy.*—Male, shoemaker, February 1, 1887.

He has been intemperate, but has not had rheumatism. During the past year he has had cough with mucous sputum and occasional vomiting.

Four months ago his legs became œdematous.

One month ago he developed ascites.

Now there is general anasarca. The urine is scanty, 1019, no albumin.

There are dulness and bronchial breathing over the lower half of the right lung. The heart sounds are feeble, but there is no murmur.

The dropsy continued and increased; he steadily lost flesh and strength. February 17 he died.

Autopsy.—The brain was not examined.

Both ventricles of the heart are dilated, without hypertrophy; the valves are normal.

The right pleural cavity is half-full of serum; the right lung is compressed. The left lung is œdematous and there is fibrin on the pleura.

The liver weighs two pounds; the capsule is thickened; there are no minute changes.

The mucous membrane of the stomach and small intestine is congested.

The kidneys are in the condition of chronic congestion.

CASE IV. *Subacute Dilatation of the Heart with Dropsy.*—Male, 27 years old, driver, February 13, 1877.

He has not been intemperate; he has not had syphilis or rheumatism. He has been in good health until five weeks ago. Then he caught cold and began to cough, with mucous sputum, occasional pain in the epigastrium, and dyspnœa on exertion, which has been constantly increasing.

Now he is fairly nourished, with good appetite. The heart's action is regular but feeble; there is no murmur. I made the diagnosis of dilatation of the ventricles, without hypertrophy or valvular lesions.

The urine is 1020, fair quantity, no albumin.

The dyspnœa persisted, he became more and more feeble, developed general dropsy, and died April 20.

Autopsy.—The brain was not examined.

There is an increase of clear serum in the pericardium. Both ventricles are dilated, without hypertrophy; the mitral valve is insufficient, but not thickened. There is a large thrombus adherent to the wall of the left ventricle.

The lungs are in the condition of pigment induration, with serum in both pleural cavities.

The liver is of normal size and hard.

The spleen is small and hard.

The stomach shows the changes of chronic gastritis.

The kidneys are in the condition of general congestion.

There is general dropsy.

CASE V. *Chronic Dilatation of the Heart with Dropsy.*—Male. 42 years old, lawyer, August 16, 1879.

He is intemperate and has had syphilis, but was in fair health until three months ago. Then he began to have dyspnœa, palpitation, vertigo, and headache.

For one month he has had œdema of the legs and a little cough.

Now he has general dropsy; urine 1010, in fair quantity, considerable albumin.

September 30 he had improved enough to go out. The improvement did not last; the dyspnœa and venous congestion came back and continued until he died on October 27.

Autopsy.—The brain was not examined.

The heart weighs one and a half pounds. Both ventricles are dilated, without hypertrophy. There is a little old thickening of the aortic and mitral valves. There are no minute changes in the walls of the ventricles.

There is serum in both pleural cavities; the lungs are in the condition of pigment induration.

The liver is large, hard, and congested.

The mucous membrane of the small intestine and stomach is congested and coated with mucus.

The kidneys weigh together twenty-four ounces; they are smooth, hard, and congested.

Case VI. *Chronic Dilatation and Hypertrophy of the Heart.*—Male, 63 years old, November 22, 1883.

For eight months he has suffered from dyspnœa and dropsy of the feet and legs.

He is now emaciated, with thickened and contracted arteries, dyspnœa, œdema of the legs, the heart enlarged but with no murmurs. The dyspnœa and dropsy continued; urine 1018, with a little albumin. He became delirious, and died suddenly December 6.

Autopsy.—The brain was not examined.

There is general subcutaneous dropsy.

Both ventricles of the heart are much dilated and moderately hypertrophied. There is a little old thickening of the aortic valves.

The lungs are in the condition of pigment induration, with serum in the pleural cavities.

The liver and spleen are hard and congested.

The mucous membrane of the stomach and small intestine is congested.

The kidneys are in the condition of chronic congestion passing into chronic nephritis.

The Fatty Heart.

1. There is an accumulation of fat beneath the pericardium. This occurs in persons who also have accumulations of fat in other parts of the body.

The heart's action is often feeble, and there is dyspnœa on exertion. It is difficult to tell whether the cardiac symptoms are due to the fat on the heart or to the general condition of the patient.

The indications for treatment are by a regulated diet and systematic exercise to remove the fat.

2. There is a fatty degeneration of the muscular fibres of the walls of the heart.

The heart remains of its normal size or becomes somewhat smaller; it may be dilated. Its walls are soft and lighter colored. The degeneration may involve the walls of both ventricles, or of one, or of part of one. The degeneration may advance so far at some one point that the wall of the ventricle will be ruptured.

Causes.—This change in the heart may be secondary to profound anæmia, to long and wasting diseases, to the severe infectious diseases, and to poisoning by phosphorus. It occurs also as a primary disease, and is then especially common in males between the ages of 40 and 70.

Symptoms.—Physical signs: The heart is of normal size, dilated or diminished. The impulse is feeble; the first sound is short and feeble; the action may be regular, irregular, fast, or slow. The systolic murmur at the apex, or at the second left interspace, which occurs without valvular lesions, may be present.

There may be pain, referred to the heart, either continuous or spasmodic; or attacks of angina pectoris.

There may be attacks of syncope, dyspnœa on exertion, or constant dyspnœa. Cerebral symptoms: vertigo, convulsions, loss of consciousness, or coma may be developed. There may be gradual loss of flesh and strength. Dropsy is not a frequent symptom.

Some of the patients never give any symptoms of their cardiac lesion, and die of some other disease.

Some give no symptoms until they have a fatal attack of angina, of syncope, of coma, or of rupture of the heart.

Some give the characteristic symptoms of the disease for months or years.

Treatment.—The best that can be done is the regulation of the diet and mode of life, and the use of cardiac stimulants.

CASE I. *Fatty Degeneration of the Heart; Dropsy; Sudden Death.*—Male, 24 years old, November 13, 1894.

Intemperate; no rheumatism; no syphilis. Four weeks ago dropsy began in the feet and extended upward until there was general subcutaneous dropsy. There was also a good deal of dyspnœa.

Now there is general subcutaneous dropsy. Breathing 17 and labored; temperature 98.6°; pulse 93, regular, no tension. Urine 1010, no albumin, 60-80 ounces daily. No physical signs over the heart or lungs.

November 16: Is sleeping well, breathing well, and the dropsy is disappearing.

November 18: While sitting quietly reading, suddenly had several convulsions, became comatose, and died.

Autopsy.—The brain, its membranes and blood vessels are normal.

The lungs are covered with old pleuritic adhesions.

The left ventricle of the heart is dilated; its wall has undergone fatty degeneration.

The liver and stomach are congested.

The spleen and intestines are normal.

The kidneys are normal to the naked eye.

CASE II. *Fatty Degeneration of the Heart; Rupture of the Left Ventricle.*—Male, 50 years old, merchant, September, 1868.

He was apparently well until three days before his death. Then he began to have moderate precordial pain lasting through the three days. Then, while sitting quietly in his chair, he suddenly died.

Autopsy.—The pericardial sac is full of blood.

There is a rupture in the wall of the left ventricle about one inch from the septum. The rupture is one-fourth of an inch long, directly through the heart wall. The heart weighs sixteen ounces. There is some old thickening of the aortic and mitral valves. The muscular fibres of the heart wall are far advanced in fatty degeneration, many of them containing oil globules of large size.

CASE III. *Fatty Degeneration of the Heart; Rupture of the Left Ventricle.*—Male, 44 years old, January 7, 1883.

On January 23 the operation of internal urethrotomy was performed.

January 24: He had chills followed by a temperature of 105°.

January 25: There was total loss of motion and sensation of the left arm and left side of the face.

January 31: Motion and sensation had returned.

March 7: The pulse was intermittent: the heart sounds were indistinct.

March 12 he died suddenly.

Autopsy.—The brain and its blood vessels are normal.

The pericardial sac is filled with fluid and clotted blood. The wall of the left ventricle is thin and yellow: near the apex is a small rupture. The wall of the left ventricle is far advanced in fatty degeneration.

The lungs are [illegible].

The kidneys contain a few white infarctions.

The stomach, spleen, and liver are normal.

CHRONIC MYOCARDITIS.

Definition.—A chronic productive inflammation of the connective-tissue stroma of the heart, resulting in a production of new connective tissue and degeneration of the muscular fibres.

Lesions.—The wall of the left ventricle, the septum between the ventricles, and the papillary muscles are the portions of the heart most frequently involved. The inflammation may be diffuse, involving most of the wall of the left ventricle, or circumscribed, involving only a part of it. The changes are usually most marked at the apex of the left ventricle. The coronary arteries and the arteries in other parts of the body regularly show the lesions of chronic arteritis. The left ventricle may be dilated or hypertrophied. There may be thrombi in the left ventricle. In the portion of the heart wall affected by the inflammation there are found a new growth of connective tissue, sometimes an infiltration with pus cells, and degeneration or atrophy of the muscular fibres.

The inflamed portion of the heart wall may rupture, or may be pouched out so as to form an aneurism.

Etiology.—In some cases the myocarditis seems to be secondary to occlusion of the coronary arteries or to an adherent pericardium. In other cases it is due to the same causes which produce chronic productive inflammations in other parts of the body.

Symptoms.—The diagnosis of myocarditis is made very frequently by physicians who are not in the habit of seeing autopsies. Whenever a patient with pericarditis or endocarditis begins to have very bad heart action, it is said that he has developed myocarditis. In the same way, if the heart's action is bad, without valvular murmurs, the diagnosis of myocarditis is ordinarily made.

As a matter of fact, chronic myocarditis is of rather rare occurrence, and the diagnosis is a difficult one.

1. There are patients with advanced myocarditis who give no pronounced symptoms and continue at their usual occupation up to the time of death. The death is usually sudden.

The most marked example of this I have seen was in a surgeon whom I saw frequently. He died in his carriage on his way home after performing a surgical operation. Nearly the entire wall of the left ventricle was changed into connective tissue. He had never had any cardiac symptoms except a rapid pulse.

2. There are patients who give the regular symptoms of inefficient heart action. These symptoms may develop slowly or come on suddenly only a few weeks before death.

The symptoms are the regular ones of venous congestion of different parts of the body:

Delirium and coma.

Cough with mucous or bloody sputum, the pneumonia of heart disease.

Vomiting and loss of nutrition.

Dropsy of the serous cavities and the subcutaneous connective tissue.

Urgent cardiac dyspnœa.

Diminution in the quantity of the urine and the presence of albumin.

The cardiac symptoms are those of dilatation of the ventricles, inefficient heart action, and sometimes a systolic murmur.

Death is often sudden.

Treatment.—This, as a rule, is unsatisfactory. We employ the different cardiac stimulants, but it is very difficult to improve the action of the heart.

CASE I. *Myocarditis; Emphysema.*—Male, 51 years old, cigarmaker. April 23, 1888.

Eighteen years ago he had a chancre. In 1868 he had a severe attack of rheumatism. In 1875 he had a second attack of rheumatism.

Six weeks ago he noticed that he had dyspnœa on exertion. After a week he took cold and had cough with mucous sputum, dyspnœa, fever, and was in bed for three weeks. Then he improved and went to work, but continued to have dyspnœa on exertion.

April 22, after breakfast, he had a sudden attack of urgent dyspnœa with vomiting.

April 23: Pulse 120, breathing 40, temperature 99°. The radial arteries are tortuous. Urine 1017, trace of albumin. Over both lungs there are the physical signs of emphysema and bronchitis. The heart is of normal size, with a systolic murmur at the apex.

He was sick for seven weeks with cough, constant hæmoptyses, dyspnœa, dropsy, and loss of flesh and strength.

Autopsy.—The brain was not examined.

There is general dropsy.

Both ventricles are dilated, with thinned walls and a growth of connective tissue between the muscular fibres. There is an old thrombus entangled in the trabeculæ of the left ventricle; the valves are normal.

The left pleura is two-thirds full of serum; the pleura is coated with fibrin. Both lungs are in the condition of pigment induration; the left lower lobe is infiltrated with blood.

The stomach, small intestine, liver, and kidneys are congested.

Case V. *Chronic Myocarditis.*—Male, 43 years old, hatter, May 5, 1879.

Intemperate; no history of rheumatism; five years ago an indurated chancre, but no secondary symptoms.

Ten weeks ago he began to cough, with mucous sputum, and to have dyspnœa and palpitation on exertion.

For three weeks he has had dropsy of the feet, nausea and vomiting, headache, and has lost flesh and strength.

Now he has urgent dyspnœa; pulse 118; general dropsy; urine 1020, albumin, twenty-two to eighty-two ounces in the twenty-four hours.

Up to May 1 he improved; the dropsy disappeared and he was out of bed. After this the urine was scanty and the dropsy returned.

June 15, after walking to the water closet, he returned, sat down, ate an orange, fell to the floor, and was dead in a few minutes.

Autopsy.—The pia mater of the brain is somewhat thickened and infiltrated with serum; the convolutions are small; the arteries are normal.

Both ventricles of the heart are dilated and hypertrophied. In the lower part of the left ventricle there is an old thrombus adherent to the endocardium. The endocardium of the left auricle and ventricle is thick and white. The aortic and mitral valves are a little thickened. Half of the thickness of a large part of the wall of the left ventricle is converted into fibrous tissue.

The right pleura is half-full of reddish serum. The pulmonary and costal pleura is congested, thickened, and covered with connective tissue. The middle and lower lobes of the right lung are compressed.

The liver and spleen are normal.

The kidneys weigh together sixteen ounces; they are hard and contain several infarctions.

RUPTURE OF THE HEART.

Lesions.—Ruptures of the heart are most common in the anterior wall of the left ventricle near the apex. They also occur in the wall of the right ventricle, the right auricle, the septum between the ventricles and the papillary muscles. The rupture is small, direct or indirect. The rupture is due to fatty degeneration of the muscle, or to occlusion of one of the branches of the coronary artery, or to myocarditis.

Symptoms.—The rupture may occur during exertion, or while the patient is perfectly quiet.

If the rupture is direct, death is almost instantaneous. If it is indirect, the patients may live for hours or days. They have sudden pain in the heart, vomiting, dyspnœa, prostration, irregular and feeble heart action.

I can recall only two cases in which I made the diagnosis of rupture of the heart twelve hours before the patient's death. I think there is something characteristic in the peculiar cardiac oppression developed in these cases.

(See Case II. under Fatty Heart.)

THE NEUROSES OF THE HEART.

The Irregular Heart.

There is a large class of cases of disturbance of the action of the heart without structural disease for which we have no good name, and which we cannot as yet subdivide in a satisfactory manner.

The heart's action may be too rapid, or irregular, or intermittent, or exaggerated, or too slow. These changes in the heart's action either occur in paroxysms, or continue for long periods; they occur after exciting causes, or continue when no cause can be discovered.

Such an irregular heart action is accompanied with consciousness of the heart's action; abnormal cardiac sensations; feelings of constriction, displacement, dropping of blood, etc.; more or less mental anxiety; vertigo, headache, fulness of the head, syncope, and sleeplessness; loss of flesh and strength; disturbances of digestion.

Many of the cases are mild and easily relieved, but others give serious symptoms and are but little benefited by treatment.

The diagnosis of many of the cases is easy; but it may be difficult to distinguish this condition from exophthalmic goitre, chronic myocarditis, disease of the coronary arteries, the fatty heart, or stenosis of one of the valves without a murmur.

The physical signs are the change in the action of the heart, and sometimes a murmur at the second left space or at the apex.

To the cases in which the heart's action is exaggerated the name of "palpitation" is often given. It can be seen and felt that the contractions of the ventricles are unnaturally forcible. The mere consciousness of the heart's action is a discomfort, and in addition there may be the feelings of pain or constriction.

In the simplest cases of palpitation the consciousness of the heart's action is the principal symptom. But there are other cases which are more complex—pain, either continuous or paroxysmal, shortness of breath, feelings of displacement or constriction, mental anxiety, cerebral symptoms, sleeplessness, loss of nutrition, and disturbances of digestion. While many cases of palpitation are mild and easily relieved, there are others that are severe, long-continued, liable to relapse, and difficult of treatment.

Causes.—Tobacco and tea are the most important causes of palpitation. They may be efficient by themselves, but their most marked results are seen when combined with over-exertion, either mental or bodily, and epidemic influenza.

The name tachycardia is given to the cases in which the heart's action is too rapid, whether it is forcible or feeble. It occurs without organic disease of the heart, or when the disease is not sufficient to account for the rapid heart action. Its causation is unknown.

Tachycardia may occur in paroxysms, or be continuous for long periods.

The paroxysms vary in their severity from mere feelings of discomfort to a condition of dyspnœa and disturbed circulation which looks as if it would prove fatal. Between the paroxysms the heart's action may be perfectly normal.

In the long-continued cases disturbances of digestion, loss of nutrition, and nervous phenomena make up a large part of the clinical picture.

The names "cardiac asthenia," or "heart exhaustion," are given to the cases in which the heart's action is too feeble, although there is no disease of the heart. The patients are often obliged to stay in bed. If they sit up they feel faint or lose consciousness. The heart's action is feeble, the pulse small and compressible, and generally increased in frequency. There may be uncomfortable feelings about the heart, but no pain. The skin is cold, the appetite poor, the bowels constipated. The patients are sleepless, nervous, and depressed. In most of these cases the cardiac symptoms are only the most prominent symptoms of the condition of neurasthenia.

Treatment.—We first try to find out if there is an exciting cause for the disturbed heart action which can be removed or treated. Such causes are tobacco, coffee, tea, disturbances of digestion, anæmia, and hysteria.

If such an exciting cause can be discovered and be removed, or successfully treated, we may be able to cure the cardiac disturbance; but it may well be that all attempts at treatment on these lines turn out to be useless, and that we have to try to directly influence the action of the heart. For this purpose we employ digitalis, convallaria, caffeine, strophanthus, potassium iodide, cannabis indica, aconite, belladonna, or opium, separately, or combined in various ways.

The general management of these patients is a matter of much importance. Some of them need rest in bed, others require graduated exercise. Much can be done by the careful use of hot and cold baths and of massage. The diet must be so regulated that the patients get enough to eat. All functional disturbances of the stomach and bowels are to be corrected.

CASE I. *Tachycardia with Attacks of Pain and Breathlessness* *and Dilatation of the Stomach.*—Male, 29 years old, polisher, November 14, 1902.

He was well and at work until eight months ago. Then he began to have attacks of pain in the epigastrium, nausea, and breathlessness. These attacks have been repeated frequently and he has lost forty pounds in weight.

Now he is emaciated; there are no physical signs over the heart or lungs; pulse 100; hæmoglobin 69 per cent.; the stomach is considerably dilated and distended with gas.

December 12: He has improved; has gained two pounds; no pain, but still has attacks of dyspnœa; hæmoglobin 75 per cent.; stomach not as much dilated.

December 26: The attacks of pain and dyspnœa have returned; pulse is 120; he has lost weight; the stomach is not dilated.

January 16, 1903: He has gained five pounds; is fairly comfortable; pulse 100.

February 2: He has had two bad attacks of pain and palpitation; pulse 110; no dilatation of the stomach.

March 21: He has been feeling better and has gone back to work, but the pulse is 125.

April 17: He has continued to feel well and to work; pulse 130.

October 10: He has continued to work; has gained a good deal in flesh and strength; has no pain; can eat well, but the pulse continues at 100.

December 8: Pulse 96; doing well.

Angina Pectoris.

It has been customary to describe angina pectoris as a disease and to speak of its lesions, causes, and symptoms. It seems to me that the time has fully come to recognize the fact that there is no such disease as angina pectoris, but that attacks of pain of greater or less severity constitute one of the symptoms of a variety of cardiac affections. We may, if we choose, call the more severe attacks of pain false angina or true angina.

1. With some of the cases of functional disturbance of the heart, attacks of pain, sometimes quite severe, may be a prominent symptom. Generally speaking, these pains belong to the class of hysterical pains, and the treatment is that of hysteria. But in some of the cases it is not easy to account for the pain or to cure it.

2. With all forms of chronic endocarditis, myocarditis, fatty

heart, hypertrophied heart, and dilated heart, there is the liability to attacks of pain, which may last for minutes, days, or weeks. The pain is more or less severe, begins in the heart and radiates down the left arm, is regularly accompanied with some disturbance of the heart's action. The pain is a real pain, due in some way to the condition of the heart. We do not know why one person has it and not another, nor what brings on the particular attacks, nor why it is that some attacks are fatal.

The management of these patients is that of all cases of organic disease of the heart, with the addition of opium and cardiac stimulants.

3. With chronic inflammation of the aortic valves, of the arch of the aorta, and of the coronary arteries, persons are liable to attacks of pain which are particularly severe and are accompanied by the feelings of cardiac oppression and of impending death. It is to these attacks that the name of angina pectoris is given.

These conditions of the heart are most common in persons over 50 years of age. The attacks are commonly brought on by contraction of the arteries.

The pain is referred to the precordial part of the lower sternal region, exceptionally to the middle or upper sternal region; with the pain there may be tenderness on pressure. The pain radiates to the mid-dorsal spine, to the left or to both sides of the neck and occiput, to the left shoulder and arm, to both arms, rarely to the right arm alone, to the left leg, or to both arms and the left leg. With the pain there may be feelings of numbness and tingling. The pain is severe and occurs in paroxysms which last for minutes or hours.

There is a feeling of cardiac oppression and a sense of impending death which produces a profound impression on the patient.

The pulse varies with the character of the attack and with the condition of the heart. During the paroxysms it is either a high-tension pulse or a feeble and irregular pulse.

The breathing also varies in the different cases. There may be real dyspnœa with cyanosis, or only a feeling of dyspnœa, or nearly natural breathing.

There may be violent and continued eructations of gas, or vomiting.

The mind usually remains clear; there may be slight or severe general convulsions; the patients may lose consciousness or they may have vertigo.

The expression of the face is anxious; the skin is cold and often bathed in perspiration.

The attacks usually come on suddenly and without warning. They may, however, be preceded by abnormal cardiac sensations, or be excited by certain muscular movements, or occur at regular intervals.

Each attack may last for a few minutes or for several hours. A patient may have only a single attack, from which he recovers, or during which he dies. He may have a number of attacks, any one of which may prove fatal; or after a number of attacks there may be no further recurrence.

The prognosis of the disease is very serious, but yet there is a considerable number of persons who suffer from one or more attacks and entirely recover.

The prognosis is rendered very difficult by the fact that inflammations of the arch of the aorta and of the coronary arteries do not necessarily give any physical signs. Also, the contraction of the arteries which bring on the attacks may occur when the organic changes in the heart are in their early stages, or when they are far advanced.

Treatment.—This is principally directed to the condition of the arteries. At the time of the attack we use nitrite of amyl, nitroglycerin, and morphine. To prevent the attacks we use chloral hydrate, the iodide of potash, and regulate the mode of life.

CASE 1. *Angina Pectoris; Sudden Death; Chronic Inflammation of the Aorta, Constricting the Openings of the Coronary Arteries.*—Male, 46 years old, wood moulder, March 28, 1886.

He is intemperate; has had syphilis, but no rheumatism.

During the past five weeks he has suffered from attacks which he describes as follows: First a feeling of oppression over the front of the chest, with increased efforts to breathe. Then a sharp pain in the left side of the chest, radiating over the front of the chest and into both shoulders and arms; with this the feeling that he is about to die.

In some attacks he belches wind, vomits, and sweats.

At first there were two or three paroxysms a day; later five or six a day, each one lasting about twenty minutes.

Now he is well nourished, pulse 96, breathing 40, temperature 98°. There are no physical signs over the heart or lungs.

Shortly after admission he began to complain of great pain in the chest, with dyspnœa, vomiting, the radial arteries hard and cord-like. After a little while the attack subsided.

May 29 he was found dead in bed.

Autopsy.—The brain was not examined.

The lungs are fully inflated, the lower lobes congested and œdematous.

The cavities of the heart are filled with red and yellow clots. The left ventricle is moderately dilated, with thin walls. The aortic valves are thickened and insufficient; the mitral valves are thickened.

There are patches of thickening through the whole length of the aorta. Just above the valves is a large, deep patch partly calcified. This involves the openings of both coronary arteries; beyond this point the arteries are normal.

The liver, spleen, and kidneys are congested.

EXOPHTHALMIC GOITRE.

Basedow's disease. Graves' disease.

The cases are characterized by rapid heart action, enlargement of the thyroid gland, and protrusion of the eyeballs.

Lesions.—After death the thyroid gland is found simply hypertrophied; there is nothing in the orbits; the left ventricle of the heart may be hypertrophied. In a few cases in the ganglia of the cervical sympathetic there have been found degeneration of the nerve cells and a new growth of connective tissue.

Causes.—The disease is much more common in women than in men, and especially during early adult life.

Fright, or severe mental shock, or over-fatigue are believed sometimes to act as causes. Rheumatism occurs as an antecedent condition in a number of cases. The association of the disease with other nervous disorders, such as epilepsy, hysteria, chorea, diabetes, and insanity, has often been noticed. In women, anæmia often precedes the disease. There is, in some families, an hereditary predisposition.

Symptoms.—The symptoms are usually developed slowly and gradually, but exceptionally are well marked within a few days.

There are three cardinal symptoms: a rapid heart action, goitre, and exophthalmos. The rapid heart action is the most constant symptom, the most important, and usually the one first developed. The heart's action is rapid, regular, forcible, and after a time attended with hypertrophy of the left ventricle.

The enlargement of the thyroid gland follows the rapid heart action; it is usually, but not always, present.

The exophthalmos is the least constant of the three symptoms, and the last to be developed.

Besides the three characteristic symptoms of the disease, there may be others.

There may be tremor affecting the muscles of the whole body or only those of the arms. It is a tremor like that which is noticed when the muscles are over-fatigued. It is more obvious when the patient is flurried, and sometimes may be noticed only under such conditions. It is more perceptible when the patient is sitting up than when lying down. It only interferes with the more delicate movements of the hands, such as writing or sewing.

There may be painful cramps of some of the muscles, especially at night.

Occasionally there are slight and temporary elevations of temperature.

Sweating is frequent and sometimes excessive.

There may be a brown pigmentation of the skin of the face and of other parts of the body.

Anæmia is very frequently present, especially in young women.

In the severe cases there is well-marked loss of flesh and strength.

Many of the patients become nervous and irritable, hypochondriacal, or hysterical. Some become fairly insane.

The course of the disease is usually protracted for years; but cases are reported which have lasted only a few months or days.

Some of the cases recover altogether, some only in part. Death is due to some intercurrent disease, to loss of nutrition, to pressure on the trachea by the thyroid tumor, to syncope, or to attacks of dyspnœa.

Treatment.—The indications are to improve the general health, to cure the anæmia, and to regulate the action of the heart. For the heart action digitalis, strophanthus, belladonna, or aconite is given, and either the galvanic or faradic current may be applied to the neck.

Good results are reported after removal of the thyroid gland.

THE ARTERIES.

Chronic Arteritis.

Synonyms.—Arterio-capillary fibrosis, arterio-sclerosis.

Definition.—A chronic productive inflammation of the walls of the arteries, which involves principally their inner and middle coats, and is often accompanied by degeneration.

Lesions.—This form of inflammation occurs most frequently in the aortic system of arteries, but it is also seen in the branches of the pulmonary artery. Only a part of the aorta is involved, or the whole of the aorta, or the aorta with most of its branches, or the arteries of some particular part of the body.

There is often chronic productive inflammation of one or more of the viscera.

Causes.—It is evident that these morbid changes are caused by lead-poisoning, alcohol, gout, and syphilis; that the disposition to them is hereditary in some families; that they constitute one of the regular senile changes; that they are often associated with chronic diseases of the viscera; that the patients can be unconscious of their existence, and that, on the other hand, they can cause most distressing symptoms, and even death. Chronic arteritis is seen so often in persons over 50 years of age, in fact but few such people have normal arteries, that we are apt to forget that it is also to be seen in young adults and children.

Thoma and others believe that the changes in the walls of the arteries are due to slowing of the blood current. They teach that:

1. Every long-continued slowing of the blood current causes contraction of the middle coat of the aorta, and, if this is not sufficient to accelerate the blood current, to a growth of connective tissue in the intima.

2. Primary diffuse and nodular arterio-sclerosis depends upon a weakening of the wall of the blood vessel due to constitutional conditions. This is followed by dilatation of the vessel, slowing of the blood stream, and then the growth of connective tissue in the intima.

3. Secondary arterio-sclerosis is caused by slowing of the blood current produced by changes of the circulation in the capillary vessels.

It appears to me that the most practical way to look at all these changes in the arteries is to consider them the results of a combination of chronic productive inflammation and degeneration occurring in connective tissue. We shall then think of the arteries as we do of the endocardium, or the liver, or the kidneys, as a definite part of the body, liable to become the seat of chronic inflammation from the same causes as those which produce similar changes in other parts of the body.

Lesions: 1. *The Small Arteries.—(a)* The simplest change in the small arteries is an increase in the size and number of the endothelial cells. This is best seen in the arteries in miliary tubercles and in the small gummata.

(b) There is a growth of new connective tissue from the endothelium, which encroaches upon the lumen of the artery and finally occludes it. The growth forms a ring on the inside of the intima, thicker in some places than in others.

(c) There is a thickening of the inner coat beneath the endothelium. The lumen of the artery is irregularly narrowed, while in other places the wall of the artery is thinned. Besides the thickening of the inner coat, the middle and outer coats may also be thickened.

(d) The whole wall of the artery is thickened and replaced by dense connective tissue.

2. *The Large Arteries.*—In the large arteries altogether the most frequent change is the thickening of the intima. This is often present in arteries which look normal to the naked eye. Besides the thickening of the intima there is often, in addition, a thickening of the middle and outer coats, or a replacement of the muscular coat by connective tissue. When all the coats are thickened in this way, the arteries often become elongated and

tortuous. Occasionally there are areas of degeneration in the thickened wall of the artery, or even infiltration with the salts of lime.

3. *The Aorta.*—The changes in the aorta differ from those in the arteries by reason of the combination of degeneration and necrosis with the growth of new tissue due to the chronic inflammation, by the frequency of calcification, and by the liability of the outer coat to purulent infiltration. We find, therefore, in the aorta:

(a) Simple thickening of the inner coat by new connective tissue.

(b) Degeneration and softening of the inner and middle coats.

(c) Calcification of the inner and middle coats.

(d) Infiltration of the outer and middle coats with pus cells.

(e) Thinning and atrophy of the inner and middle coats.

(f) The formation of thrombi on the roughened surface of the inner coat.

Symptoms.—It must be admitted that we find the lesions of chronic arteritis far advanced in persons who have never given symptoms referable to the arteries. It must also be admitted that chronic disease of the lungs, heart, liver, or kidneys is so often associated with chronic arteritis as to obscure the clinical picture. But, allowing for all these, there remains a large class of cases in which chronic arteritis is the disease.

1. Chronic inflammation of the cerebral arteries. The cerebral symptoms belonging to the inflammation of these arteries have already been described.

2. Chronic inflammation of the medium-sized and smaller arteries throughout the body. At first for a number of years the patients only suffer from impaired nutrition, a disposition to become anæmic, and attacks of dyspnœa. It can be seen and felt that the walls of the temporal and radial arteries are thickened and that the left ventricle of the heart is hypertrophied. At the times when the patient has dyspnœa the tension of the pulse is much increased.

For a considerable length of time the nutrition and the anæmia can be improved by climate and by diet. The attacks of dyspnœa can be controlled by the drugs which dilate the arteries. But sooner or later the patients get worse. Some of them get up a dyspnœa that cannot be controlled, the action of the hypertrophied heart fails, and the patients, after suffering for weeks or months with the most distressing dyspnœa, die. In other cases, death takes place with cerebral symptoms—sudden unconsciousness, aphasia, or hemiplegia. Cases of this character are of ordinary occurrence. Much less common are the cases in which the symptoms run a short and rather acute course. For example, in the "Transactions of the London Pathological Society for 1892" we find the following history:

A girl, aged 11, in the summer of 1891 had a week's illness, probably to be attributed to infarctions of the lung. In January, 1892, similar symptoms came on, the pain being first in the left side, later in the right; the cough, with blood-stained sputum, persisted up to the end of the illness. On February 7 the legs became œdematous, and a few days later the face. It was supposed that she was suffering from acute nephritis. The urine contained a little albumin and was very much reduced in quantity. The child seemed to die of uræmic poisoning. At the autopsy all the arteries of both the aortic and pulmonary systems were found to be diseased. There was consolidation with infarctions of portions of both lungs. Both renal arteries were occluded by thrombi. There were small clots in each of the lateral lobes of the cerebellum. There was hypertrophy and dilatation of the left ventricle of the heart.

3. Chronic inflammation of the femoral arteries and their branches causes gangrene of the toes or of the legs.

4. Chronic inflammation of the aorta just above the aortic valves may narrow the coronary arteries and cause attacks of angina pectoris.

5. If the whole of the arch of the aorta, with more or less of the rest of the aorta, is involved, the patients may have symptoms which resemble those of organic heart disease.

The symptoms come on in attacks; the intervals between the attacks are shorter as the disease advances. The cases vary as to the character of the attacks.

In some patients there is dyspnœa on exertion, or spasmodic dyspnœa. The spasmodic dyspnœa is not attended with any evidence of contraction of the bronchi or of the arteries.

In others there are attacks of pain like those of true or of false angina.

In others there are repeated attacks of epigastric pain and vomiting. ✓

Or there may be congestion and œdema of the lungs, or chronic congestion of the kidneys, or general dropsy.

The nutrition of the patients gradually suffers and they lose flesh and strength. The ventricles of the heart are often dilated, or hypertrophied, or both. There may be a systolic murmur, or a double murmur, and dulness on percussion over the arch of the aorta.

The natural course of the disease is to grow steadily worse and to terminate fatally, but yet for a time the symptoms are not continuous, but come on in attacks.

6. The inflammation of the aorta may be accompanied with the formation of thrombi on its roughened inner surface.

Large thrombi in the arch of the aorta may cause urgent dyspnœa, cyanosis, vomiting, prostration, absence of the heart sounds and of arterial pulsation, and death within a few days.

pressure on
ab. end

Large thrombi in the abdominal aorta produce pain, tenderness, and loss of power in the legs, pain and paralysis of the rectum, and finally gangrene of the legs. (See Case XVII. under Endocarditis.)

Fragments of such thrombi may be detached and lodged as emboli in the arteries of the brain or of the extremities.

Case I. *Chronic Inflammation and Dilatation of the Arch of the Aorta.*—Male, 28 years old, September 5, 1874.

For six months he has complained of pain in the epigastrium after eating or walking.

For fifteen days he has had severe colicky pains in the abdomen, nausea and vomiting, and became jaundiced.

September 10 he began to have urgent dyspnœa. There was a mitral systolic murmur.

September 17 he became delirious.

September 21 he died.

Autopsy.—The brain was not examined.

The left ventricle of the heart is dilated; the mitral opening is too large.

The arch of the aorta is dilated and atheromatous.

There is fluid in both pleural cavities: the lungs are in the condition of pigment induration.

The liver is small, hard, and finely nodular.

The kidneys are in the condition of chronic congestion.

CASE II. *Chronic Inflammation and Dilatation of the Arch of the Aorta with Attacks of Vomiting.*—Male, 50 years old, November 25, 1874.

For eight months he is said to have had subacute rheumatism.

Three months ago he began to be seized regularly once a week with vomiting of a thin, greenish fluid: he also had occasional cardiac palpitation and dyspnœa.

Now he is well nourished; pulse and temperature normal: urine 1022, no albumin.

December 3: An attack of profuse vomiting, during which the pulse remained good.

December 11, 17, 24, 31, and January 7: He had similar attacks of vomiting with no cardiac symptoms. There is a systolic murmur at the base of the heart.

January 16: Vomiting with severe epigastric pain and dyspnœa.

January 18 he died with a feeble heart.

Autopsy.—The brain was not examined.

The heart is normal.

The arch of the aorta is dilated and atheromatous.

The lungs, liver, stomach, and kidneys are normal.

CASE III. *Chronic Inflammation of the Aorta and its Large Branches: Symptoms of Aortic Insufficiency.*—Male, 44 years old, fireman, August 14, 1888.

Twelve years ago he had a chancre.

For ten years he has complained of frequent attacks of pain in the right side of the chest and the right lumbar region.

Eight years ago the right sciatic nerve was stretched for sciatica.

For one year he has suffered from dyspnœa and cardiac palpitation on exertion.

Ten weeks ago dropsy began in the feet and has extended upward. The dyspnœa and palpitation have become much worse.

Now he is pale, temperature 99°, breathing 30, pulse 90 and too full.

There is fluid in both pleural cavities. The heart apex is in the sixth interspace five inches from the median line; there is a loud double murmur over the whole heart. Urine 1026, trace of albumin.

The dyspnœa and dropsy continued; he became delirious and died three weeks later.

Autopsy.—There is general subcutaneous dropsy.

Both pleural cavities are half-full of serum. The lungs are emphysematous and œdematous.

There are old adhesions over the right ventricle. Both ventricles are dilated, the left hypertrophied. There is a little old thickening of the aortic, mitral, and tricuspid valves. The aortic valve is made insufficient by the dilatation of the aorta.

The whole length of the aorta and the large arteries are much changed. There is a general thinning and dilatation, with patches of thickening, some of them calcified.

The liver and spleen are large and congested.

The stomach and small intestine are congested and coated with mucus.

The kidneys are large, smooth, and congested.

Case IV. *Chronic Inflammation and Dilatation of the Aorta.*—Male, 44 years old, coachman, January 1, 1881.

Twenty-five years ago he had a soft chancre.

Four years ago he began to have pain in the epigastrium and cardiac palpitation.

Five weeks ago he began to have severe attacks of dyspnœa, coming on at night.

Now he is feeble, emaciated, and anæmic. Pulse 104, breathing 26, temperature 98°. The apex of the heart is in the sixth space to the left of the nipple; the area of heart dulness extends up to the second rib; there is well-marked pulsation in the fourth interspace to the left of the sternum. There is a double murmur at the base of the heart.

I supposed that there was an aneurism of the arch of the aorta projecting downward over the heart.

January 28 he died suddenly.

Autopsy.—The brain was not examined.

There is no pericardial effusion. Both ventricles are much dilated and moderately hypertrophied, the base of the heart extending up to the first rib. The aortic valves are thickened and insufficient.

The aorta for its entire length is dilated and its inner surface roughened; the dilatation is most marked in the arch.

The left pleural cavity is half-full of serum. The lungs are in the condition of pigment induration.

The liver is large and congested.

The spleen is normal.

The stomach is congested.

The kidneys are in the condition of chronic congestion.

CASE V. *Chronic Inflammation of the Aorta and the Large Arteries.*—Male, 43 years old, September 29, 1889.

For a number of years he has had dyspnœa on exertion and attacks of asthma.

Lately the dyspnœa has increased, and since May he has not been able to lie down.

For the past month there have been vomiting and dropsy of the feet. During the past six months he has lost forty pounds.

Now there is constant dyspnœa with exacerbations. All the arteries that can be felt are tortuous, with thickened walls and increased tension. Urine 1007, much albumin. The heart is enlarged; the lungs are emphysematous. Sometimes there is sibilant and sonorous breathing.

He continued to have an urgent dyspnœa, which could not be relieved by any drugs, and died December 22.

Autopsy.—The right pleural cavity is distended with clear serum. Both lungs are emphysematous.

The left ventricle of the heart is dilated and hypertrophied. There is some old thickening of the aortic and mitral valves. The coronary arteries are thickened.

The whole length of the aorta and all its large branches show the regular lesions of chronic inflammation.

The liver and spleen are congested.

The kidneys show the lesions of chronic nephritis.

CASE VI. *Thrombosis of the Aorta with only One Patch of Thickening.*—Male, 45 years old, October 30, 1885.

He was brought to the hospital unconscious, having fallen suddenly.

Now his face is flushed; skin warm; pupils normal; both eyes turned to the right; mouth drawn to the right; complete loss of motion and sensation, and exaggerated reflexes on the left side; aphasia; pulse 96 and full, temperature 98°. Two days later he became comatose; temperature 103°; died.

Autopsy.—The main trunk of the right middle cerebral artery is filled with a firm thrombus. The trunk of the left middle cerebral and its large branches are also filled with a thrombus, which extends into the internal carotid. The corpus callosum, fornix, right corpus striatum, optic thalamus, and internal capsule are softened.

The right internal carotid is filled with a large thrombus.

The lungs are congested and œdematous.

The heart is normal.

In the transverse portion of the arch of the aorta, just at the entrance of the innominate artery, is a large, soft thrombus adherent to the wall of the aorta. There is a patch of thickening of the inner coat of the aorta beneath this; otherwise the aorta is normal.

The liver is congested.

The spleen is normal.

The kidneys show the lesions of chronic nephritis.

CASE VII. *Chronic Inflammation of the Aorta; Death from a Thrombus just above the Aortic Valves.*—Female, 40 years old, December 24, 1879.

She was a large, fat woman, with no history except that she had been suffering from dyspnœa for several days.

She came into the hospital with urgent dyspnœa, cyanosis, constant vomiting, prostration, no radial pulse, no appreciable heart sounds.

The next day she died.

Autopsy.—The brain was not examined.

The lungs are œdematous.

The left ventricle of the heart is dilated.

The aorta for its entire length is studded with large patches of thickening. Thrombi adhere to some of these patches. Just above the aortic valves a thrombus has formed, adherent to the wall of the aorta and large enough to fill it.

The liver, stomach, and small intestine are congested.

The kidneys show the lesions of chronic nephritis.

ANEURISMS OF THE AORTA.

Fusiform and sacculated aneurisms occur either separately or together. The fusiform dilatations may involve the arch alone or the whole aorta. The sacculated aneurisms have a small or a large opening. The sacs themselves are small or reach a large size; they are either single or multiple.

Causes.—Aneurisms of the aorta are especially common in males between the ages of 30 and 50 years, in persons who have constitutional syphilis; in those who have chronic inflammation of the aorta; and in those whose occupation involves severe muscular exertion.

Situation.—Gibson gives the seat of 860 cases of aneurism of the aorta as follows:

Of the arch of the aorta, 480.

At the sinuses of Valsalva, 87.

Of the ascending portion of the arch, 141.

Of the transverse portion of the arch, 120.

Of the ascending and transverse portions of the arch together, 112.

Of the descending portion of the arch, 72.

Of the transverse and descending portions of the arch together, 20.

Of the whole arch, 28.

Of the thoracic aorta, 71.

Of the abdominal aorta at the cœliac axis, 131.

Of the lower part of the abdominal aorta, 26.

Symptoms.—These are due to the presence of the aneurismal tumor, to its pressure on the surrounding parts, and to its effect on the circulation of the blood. The presence of the tumor is more or less easily detected, according to its size and position.

The large tumors push out the wall of the chest or of the abdomen, can be seen and felt to pulsate, and give a double murmur and a thrill. Smaller tumors within the thorax only give dulness on percussion, with sometimes in addition a double murmur. Very small and deeply situated aneurisms give no physical signs.

Fusiform aneurisms can hardly be said to give a tumor in the thorax. They may give dulness on percussion and a systolic murmur. In the abdomen the dilated artery can be felt, its exaggerated pulsation recognized, and a systolic murmur heard over it.

The aneurisms are for the most part so situated that they press upon the adjacent tissues and so produce a variety of symptoms. Pressure on the descending vena cava causes venous congestion of the head and neck. Pressure on the pulmonary artery, on the right auricle, and on the upper part of the ventricles interferes with the action of the heart and causes congestion of the lungs. Pressure on the large bronchi and on the trachea sets up inflammation of their walls and mucous membranes. This inflammation often extends up the trachea to the larynx and produces cough and dyspnœa, or down the bronchi into the lungs and sets up a broncho-pneumonia. The pressure also directly produces dyspnœa and tubular breathing which can be heard over the lungs. Pressure on the œsophagus causes difficulty in swallowing. Pressure on the recurrent laryngeal nerve produces paralysis of one of the vocal cords. Pressure on the brachial plexus of nerves produces pain. Pressure on the vertebral column regularly causes erosion of the bones with great pain.

The aneurisms and the accompanying inflammation of the aorta may cause venous congestion throughout the body, and dilatation and hypertrophy of the left ventricle of the heart.

All sacculated aneurisms are liable to rupture.

Aneurisms at the Sinuses of Valsalva.

These aneurisms are sacculated, and are most frequently given off from the right sinus. Small aneurisms in this situation give no symptoms until the time comes when they rupture within the pericardium and cause sudden death. Larger aneurisms may compress and erode the pulmonary artery, the right auricle, the right ventricle, or the descending vena cava.

Corresponding to the seat of the aneurism there is an area of dulness or a pulsating tumor, either to the right or left of the sternum, at about the third interspace.

There is a murmur in thirty-four per cent. of the cases.

Dyspnœa in thirty-eight per cent.

Cough in twenty-four per cent.

Pain in twenty-one per cent.

Enlarged heart in twenty-nine per cent.

Rupture of the aneurism within the pericardium in forty-eight per cent.

Into the pulmonary artery in thirteen and five-tenths per cent.

Into the right auricle in eight and five-tenths per cent.

Into the right ventricle in five per cent.

Into the left ventricle in five per cent.

Aneurisms of the Ascending Portion of the Arch of the Aorta.

These aneurisms are sacculated in fifty-one per cent., fusiform in thirty-seven per cent. of the cases. The ascending portion of the arch of the aorta rests upon the pulmonary artery and the left side of the trachea. On its right side is the superior vena cava; on its left side the pulmonary artery. Most of the sacculated aneurisms are given off from the anterior and right aspects of the wall of the aorta, and a comparatively small number from the posterior and left aspects.

They give an area of dulness or a pulsating tumor to the right of the sternum, at the second interspace; much less frequently on the left side of the sternum. They may compress the right lung, the vena cava, the left lung, the trachea, the œsophagus, the pulmonary artery, the right bronchus, or the left bronchus.

A murmur is present in twenty-three per cent. of the cases, dyspnœa in fifty-one per cent., cough in thirty-six per cent., stridor in four per cent., pain in the chest in twenty-nine per cent., pain in the neck and shoulders in ten per cent., pain in the right arm in two per cent.

The patients remain in good health and without symptoms up to the time of their death in eleven per cent. of the cases.

Rupture of the aneurism occurs in fifty-seven per cent. of the cases. The rupture is through the wall of the thorax in eight per cent. of the cases, into the pericardial sac in twenty-two per cent., into the pulmonary artery in four per cent., into the right auricle in one per cent., into the descending vena cava in five per cent., into the trachea in one per cent., into the right bronchus in three per cent., into the right lung in five per cent., into the left lung in two per cent., into the right pleura in two per cent., into the left pleura in four per cent.

Aneurisms of the Transverse Portion of the Arch of the Aorta.

The transverse portion of the arch passes backward and to the left, over the left bronchus. The left recurrent laryngeal nerve passes behind and against this portion of the arch. The carotid, subclavian, and innominate arteries are given off from it.

These aneurisms are most frequently sacculated. The larger number are given off from the posterior wall of the aorta, a smaller number from the anterior or upper wall.

Those given off from the posterior wall soon compress the trachea, or bronchi, or œsophagus, and the recurrent laryngeal nerve. The patients very early have dyspnœa, cough, stridor, paralysis of one of the vocal cords, tubular breathing over one or both lungs, but the physical signs of the tumor are absent or obscure until it reaches a large size. These aneurisms are apt to rupture into a bronchus, into the trachea, or into the œsophagus.

The aneurisms given off from the anterior and upper wall soon give the physical signs of a tumor. They partly occlude the large arteries given off from this part of the aorta, and diminish the pulsation of the carotid and radial arteries. They must, however, attain a considerable size before they can exert much pressure on surrounding parts.

A pulsating tumor at or above the level of the manubrium is found beneath the sternum in nineteen per cent. of the cases; to the right of the sternum in twelve and five-tenths per cent.; to the left of the sternum in twelve and five-tenths per cent.

A murmur is present in fifteen per cent. of the cases, dyspnœa in seventy-one per cent., cough in fifty-seven per cent., hæmoptysis in nineteen per cent., stridor in forty-seven and five-tenths per cent., dysphagia in thirty-one per cent., pain in thirty-six per cent., weakening of one radial pulse in twenty-six per cent.

The aneurisms rupture externally in six per cent. of the cases; into the pericardium in two per cent., into the right pleura in two per cent., into the left bronchus in four per cent., into the left pleura in five per cent., into the œsophagus in six per cent., into the pulmonary artery in one per cent., into the descending vena cava in one per cent., into the trachea in ten per cent.

CASE I. *Aneurism of the Transverse Portion of the Arch of the Aorta with Inflammation of the Trachea and Spasmodic Dyspnœa.*—Female, 45 years old, December 13, 1879.

For several months she has had feelings of oppression about the chest, and during the past months has had attacks of severe spasmodic dyspnœa, coming on at night. She came into the hospital during an attack.

She sits up in bed, using all the respiratory muscles, with anxious face, cyanotic skin, finger ends clubbed. The air can be heard to enter and leave the lungs freely; there are coarse râles. She looked to me like a person in a bad attack of bronchial asthma.

December 18 she had another attack of spasmodic dyspnœa, during which she died.

Autopsy.—The brain was not examined.

The pericardial sac contains turbid serum. The aortic and mitral valves are a little thickened; the left ventricle is hypertrophied.

On the upper and posterior aspect of the transverse portion of the arch of the aorta is an aneurism the size of a hen's egg, which presses on the trachea.

The trachea is not compressed by the aneurism, but its wall is intensely congested and thickened. The congestion and thickening extend above and below the point of contact.

The lungs are covered with old adhesions, emphysematous, the bronchi congested and thickened.

The liver is congested.

CASE II. *Aneurism of the Transverse Portion of the Arch of the Aorta Opening into the Left Bronchus.*—Female, 25 years old, March 25, 1871.

Six weeks ago she began to cough, with mucous and bloody sputum, and to have dyspnœa.

For three weeks she has had afternoon fever and sweating.

Now over the lower part of the left lung there are flatness on percussion and absence of voice and breathing. Over the upper lobe there is bronchial voice and breathing.

March 29 there was a sudden large bleeding from the mouth, followed by death in a few minutes.

Autopsy.—The brain was not examined.

On the posterior aspect of the transverse portion of the arch of the aorta there is an aneurismal sac one and a half inches in diameter, nearly empty. The sac has eroded and opened into the left bronchus just below the trachea.

The left pleural cavity is half-full of turbid serum. The bronchi of the left lung are congested, thickened, and coated with muco-pus. The whole lung is thickly studded with areas of red, gray, hemorrhagic and cheesy hepatization. Some of the cheesy areas have softened.

The right lung, liver, spleen, and kidneys are normal.

Aneurisms of Both the Ascending and Transverse Portions of the Arch of the Aorta.

These aneurisms are fusiform in eighty-five per cent. of the cases. Most of them soon reach a considerable size. There is dulness on percussion, or a pulsating tumor, most frequently on the right side of the sternum, often beneath the sternum, less often on the left side of the sternum.

The pressure of the tumor presses forward and erodes the sternum and the ribs. The trachea is compressed in thirty-three per cent. of the cases, the superior vena cava in ten per cent., the œsophagus in nine per cent.

Dyspnœa is present in seventy-four per cent. of the cases, cough in forty-seven per cent., hæmoptysis in ten per cent., stridor in seventeen per cent., dysphagia in twenty-one per cent., pain in the chest in thirty per cent., pain in the back in ten per cent., pain in the arms in eighteen per cent., pain in the neck in four per cent.

The right pulse is feeble in six per cent. of the cases, the left pulse in three per cent.

The aneurisms rupture into the pericardium in ten per cent. of the cases, into the pulmonary artery in one and five-tenths per cent., into the vena cava in four per cent., into the trachea in four per cent., into the right pleura in three per cent., into the left pleura in one per cent.

Aneurisms of the Descending Portion of the Arch of the Aorta.

The descending portion of the arch lies at first on the left lung and the œsophagus, and at last rests on the left half of the body of the fifth or sixth dorsal vertebra, where the œsophagus is on the right side of the aorta. In front of it are the left bronchus and the pulmonary artery.

The aneurisms are sacculated in eighty-one per cent. of the cases, fusiform in eight per cent.

The aneurisms are given off from the posterior and left walls of the aorta.

The vertebræ are eroded in forty-two per cent. of the cases, the left ribs in front in four per cent., the trachea is compressed in twelve and five-tenths per cent. of the cases, the left bronchus in thirty-seven and five-tenths per cent., the œsophagus in thirty-one per cent., the left lung in forty-eight per cent., the right lung in six per cent.

The presence of the aneurismal tumor cannot usually be made out by physical signs until it has reached a considerable size.

A murmur is present in twenty-three per cent. of the cases, dyspnœa in fifty per cent., cough in forty-six per cent., stridor in twenty-five per cent., dysphagia in thirty-three per cent., pain in the chest in fifty-two per cent.

The aneurisms rupture into the left pleura in twenty-three per cent. of the cases, into the right lung in six per cent., into the right pleura in twelve per cent., into the trachea in four per cent., into the left lung in four per cent.

Aneurisms of the Thoracic Aorta.

These are sacculated in thirty-five per cent. of the cases, fusiform in twenty-eight per cent.

Erosion of the vertebræ occurs in seventy-four per cent. of the cases. The œsophagus, the left lung, or the right lung may be compressed, the heart may be displaced forward, there may be a pulsating tumor in the dorsal or axillary regions.

A murmur is present in four per cent. of the cases, dyspnœa in twenty-six per cent., cough in twenty-four per cent., dysphagia in nine per cent., pain in the back in forty-three per cent., in the chest in twenty-nine per cent., in the abdomen in thirteen per cent., in the left lumbar region in nineteen per cent.

The aneurisms rupture into the trachea in two per cent. of the cases, into the left lung in thirteen per cent., into the left pleura in twenty-six per cent., into the right lung in six per cent., into the right pleura in thirteen per cent.

CASE III. *Aneurism of the Thoracic Aorta with Pain in the Lumbar Region.*—Male, 33 years old, painter, July 29, 1873.

Four years ago he began to have pain in the lumbar region.

This pain has continued ever since and has been so severe that he has taken opium steadily.

Now he is emaciated, with constant intense pain low down in the back. Careful and repeated examinations gave no physical signs.

August 3 he died.

Autopsy.—The brain and its membranes are normal.

The heart is normal.

The lungs and kidneys are congested.

The liver and spleen are normal.

On the aorta are two aneurisms. The upper one, on the thoracic aorta, extends up to the third intercostal space behind; it is on the right side of the aorta, as large as a man's fist; the posterior wall of the sac is destroyed and the vertebræ are eroded.

Of the lower sac, the lower end is just below the diaphragm. Its anterior wall is hardly raised above the level of the normal aorta. The vertebræ are deeply eroded.

Aneurisms of the Abdominal Aorta.

These are usually sacculated. The larger number are situated near the cœliac axis. They are given off with about equal frequency from the anterior and posterior walls of the aorta.

Those on the posterior walls very early erode the vertebræ. They give pain referred to the vertebræ and radiating down. While the tumors remain of small size it is often difficult to make them out.

The anterior aneurisms give pain referred to the epigastrium and the anterior portion of the abdomen. The tumors are easily made out by palpation.

These aneurisms rupture into the peritoneal cavity, behind the peritoneum, into the mesentery, and into the right or left pleural cavity. The blood may escape at once into the peritoneal cavity and cause death, or it may for a time be held in behind the peritoneum and a false aneurism be thus formed.

There may be incomplete or complete obstruction of the aorta by thrombi extending from the aneurismal sac.

Treatment.—The medical treatment of aneurism of the aorta consists in determining for each patient, experimentally, whether he is better for rest or for regulated exercise, and in the administration of the iodide of potash. The iodide is given in doses of from five to twenty grains, three or four times a day. In favorable cases the aneurism is diminished in size, the symptoms are alleviated, and life prolonged.

LECTURES

ON

THE PRACTICE OF MEDICINE

WITH CASES AND CHARTS

BY

FRANCIS DELAFIELD, M.D., LL.D. (Yale)

PART IV

THE KIDNEYS

For Sale by J. T. DOUGHERTY
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NEW YORK

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BY

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THE KIDNEYS.

The kidneys are deeply seated in the lumbar region, lying one on each side of the vertebral column, at the back part of the abdominal cavity and behind the peritoneum. They are on a level with the last dorsal and the two or three upper lumbar vertebræ, the right kidney being a little lower than the left.

Dr. Brewer (*Med. News,* July 31, 1897) gives the following figures as to the position of the upper border of the kidney:

Post-mortem Examinations.—Total number of subjects examined, 151. Of these, 91 were males, 56 females, and in four cases the sex was not recorded. The upper border of the right kidney was found opposite the eleventh rib in 78 cases, opposite the twelfth rib in 62 cases, one-half inch below the twelfth rib in 1 case, and one inch below the twelfth rib in 8 cases. The right kidney had been removed in 1 case. The upper border of the left kidney was found opposite the tenth rib in one case, opposite the eleventh rib in 100 cases, opposite the twelfth rib in 43 cases, one-half inch below the twelfth rib in 1 case, and 1 inch below the twelfth rib in 2 cases. The left kidney was congenitally absent in 1 case.

The kidneys measure about 4 inches in length, 2½ inches in breadth, and 1¼ inches or more in thickness. The left is usually longer and narrower than the right. The weight of each kidney is usually stated to be about 4½ ounces in the male, and somewhat less in the female.

It is difficult to determine changes in the size and position of the kidney, unless the enlargement is considerable or the displacement marked.

In order to map out the kidneys by percussion the patient must lie on his belly; auscultatory percussion is preferable to simple percussion. We mark a point 3½ inches outward from the spines of the vertebræ at the level of the eleventh rib on either side, then measure downward from this point 4 inches and mark a second point, and then join these two points by a space 2½ inches wide. Over the central portions of the area thus included we may expect to get a change in the percussion note due to the presence of the kidney. It must be admitted, however, that it is very difficult to reach certain results by percussion alone.

Palpation is used with the patient lying on his back, side or abdomen. Any considerable enlargement or displacement of the kidney can be made out in this way. Enlarged kidneys, as a rule, are not movable by palpation nor by the movements of the diaphragm. The colon is, as a rule, pressed forward by the enlarged kidney.

Congenital Malformations.

These are of more importance to the surgeon than to the physician.

Absence of one kidney, the horseshoe kidney, the malpositions of the kidney are of frequent occurrence, and must be constantly reckoned with in operations on these organs.

The Movable Kidney.

It not infrequently happens that one, or sometimes both, kidneys cease to be imbedded in the fat of the lumbar region and become capable of a liberty of motion which increases as time goes on. The kidney continues to be fastened by its large blood vessels, and pushes the peritoneum in front of it.

It is believed that such a movable condition of the kidney may be either congenital or acquired. It occurs more frequently in women, in women who have borne children, among the laboring classes, and in persons between the ages of twenty-five and forty years. The right kidney is the one most frequently affected, next the left kidney, least often both kidneys.

Symptoms.—We can only be certain of the existence of a movable kidney when we are able to distinctly feel it. It feels like a solid tumor, not as hard as the spleen, of the shape of the kidney, movable, easily escaping from the hand which tries to grasp it, easily pushed back into its natural position. In order to make out such a kidney it is usually necessary to put the patient into different positions, so as to displace the organs; it is often necessary to examine on several different days before we can be sure of it.

Such a movable condition of the kidney may never cause any discomfort, and the patient may remain entirely unconscious of its existence. In other cases, however, the abnormal position of the kidney may give rise to a variety of symptoms.

1. In a considerable number of cases the movable kidney seems to be the cause of gastric symptoms—loss of appetite, pain, nausea, and vomiting. These gastric symptoms are not necessarily constant, but may come and go although the kidney remains movable.

2. In some cases the prominent symptom is pain in the back. This pain is referred to the side on which the kidney is displaced. It is a severe pain, made worse by standing and walking.

3. The patients discover themselves, or are told by a physician, that they have an abdominal tumor. This naturally fills the patient with alarm, and so may give rise to a variety of nervous symptoms for which the displacement of the kidney can hardly be held responsible.

4. Less frequently the symptoms are more severe. There are feelings of dragging, of pressure, or of weight in the abdomen. There may be nausea, vomiting, and pain in the abdomen. Intense pains may radiate toward the epigastrium, the sacral and lumbar region, the intercostal spaces, the shoulders, along the ureter, into the testicle or the labia majora.

It is said that there may be attacks of localized peritonitis around the displaced kidney, with chills, fever, intense pain, and hardness and tenderness of the abdomen.

The pelvis of the displaced kidney may become inflamed, or may contain calculi.

By the bending of the ureter there may be produced either temporary or permanent hydronephrosis.

91cd

Treatment.—The number of patients in whom the kidney is more or less movable is very large. In persons who complain of gastric or intestinal symptoms, or of pain in the abdomen and back, we examine for a movable kidney as a matter of routine. Experience teaches us that the symptoms may disappear although the kidney remains movable; the symptoms may disappear with treatment directed to the stomach, the intestines or the general health; the symptoms may return after the kidney has been fastened in its place by a surgical operation.

The rules for treatment are:

✓ Never tell a patient unnecessarily that the kidney is movable.

To try carefully what can be done by treatment of the stomach and intestines, by diet, massage, regulated exercise, or the rest cure.

To be sure that abdominal supports, binders and pads really make the patient more comfortable instead of less so.

To employ surgical measures, such as the suture, or removal of the kidney, only in cases in which the symptoms are distressing and other means have failed.

THE URINE.

QUANTITY.

In healthy adults consuming the ordinary quantities of fluids and solids the daily discharge of urine is 1,250 c.c., or 50 fluid ounces, or 3 pints. This quantity is liable to a considerable amount of variation according to the quantity of fluid taken and the amount of perspiration.

Complete occlusion of the pelves of the kidney or of their ureters leads to complete suppression of urine. It is not that urine is formed and cannot escape on account of the occlusion, but that the kidneys cease to perform their functions. Suppression of urine is always fatal, but yet can be borne for a number of days almost without symptoms. Sooner or later, however, prostration, delirium, stupor, and the typhoid state are developed.

Severe injuries and surgical operations, especially those on the urethra and bladder, may be followed by a suppression of urine which is often fatal. It is probable that this suppression is due to an acute congestion of the kidneys. Any disturbance of the circulation which produces either acute or chronic congestion of the kidneys regularly diminishes the quantity of urine. So we find that in acute and chronic congestion of the kidney, in acute nephritis, in the exacerbations of chronic nephritis, and in attacks of contraction of the arteries the quantity of urine is notably diminished.

When the body temperature is considerably higher than the normal the urine is diminished in quantity.

In saccharine diabetes patients pass very large quantities of urine, the kidneys being apparently excited to increased activity by changes in the composition of the blood. In cases of insipid diabetes the quantity of urine of low specific gravity is large, but it is not determined why this increase takes place.

In the slow forms of chronic nephritis, whether with or without exudation, it is the rule to have increased quantities of urine of low specific gravity, the quantity being especially large and the specific gravity very low if the vessels are the seat of waxy degeneration. But this increased production of urine may at any time be checked by changes in the heart's action, by contraction of the arteries, or by an exacerbation of the nephritis.

SPECIFIC GRAVITY.

The determination of the specific gravity of the urine gives us the relative quantity of its solid and fluid constituents. To obtain practical information on this point it is necessary to examine the urine passed at different times in the day on a number of days. In healthy persons and under ordinary conditions, the specific gravity ought not to vary much from 1.020. It seems to be generally agreed that the solid portions of the urine are excreted by the epithelium of the convoluted tubes and the fluid portions filtered through the Malpighian bodies. We would expect, therefore, that a diminution in the specific gravity would be caused by changes in the renal epithelium, and the diminution in the quantity of the urine by atrophy of the Malpighian bodies. As a matter of fact, the kidneys behave differently. When the morbid changes are confined to the epithelium, as in acute and chronic degeneration, the specific gravity is not lowered; when the Malpighian bodies are atrophied in chronic nephritis the quantity of urine is not necessarily diminished. A persistent low specific gravity means a chronic nephritis with a large production of new interstitial connective tissue, or with waxy degeneration of the blood vessels; or it means insipid diabetes. In chronic nephritis the specific gravity remains low, even if the quantity of urine is very much diminished. But in insipid diabetes the specific gravity rises as the urine is diminished in quantity.

An increase in the specific gravity regularly accompanies saccharine diabetes and chronic congestion of the kidneys.

CONSTITUENTS, NORMAL AND ADVENTITIOUS.

Urea.—The most important of the solid constituents of the urine is urea, of which a healthy adult excretes every day about 500 grains. While the specific gravity of the urine gives a general idea of the relative quantity of urea, yet there are sources of error. It is best to get the whole daily excretion of urea for several days by the hypobromite method. The principal importance of this is in determining the prognosis of cases of chronic nephritis. When the daily excretion of urea is much below the normal the prognosis is bad, although the patients may seem to be doing well.

It is also a help in distinguishing between acute and chronic nephritis, as the quantity of urea is not much affected in acute nephritis.

If, however, we examine the urine for urea as a matter of routine in every case of kidney disease we meet with many surprises and exceptions, and we learn to think of changes in the formation of urea as well as changes in its excretion. We see persons who for years do not excrete more than 200 grains of urea a day and yet enjoy perfectly good health. We see others with contracted arteries and renal dyspnœa, who are excreting more than 500 grains a day.

Urates, Oxalates, and Phosphates.—The presence of an excess of uric acid, of the urates, of oxalate of lime, and of the phosphates are of importance, not because they indicate disease or disturbance of the functions of the kidneys, but because they show disordered digestion and an abnormal condition of the blood. There are many cases of kidney disease in which the treatment of these disturbances is of the greatest importance.

Blood.—Hæmaturia is an evidence of bleeding from some part of the genito-urinary tract. So far as the kidneys are concerned the blood comes from their pelves, or from the kidneys themselves. Bleeding from the pelves occurs with pyelitis, with calculi in the pelvis, and with new growths of the pelvis. Bleeding from the kidney itself is found in acute nephritis, with exacerbations of chronic nephritis, with tubercular nephritis, with the hemorrhagic forms of the infectious diseases, and with malignant growths.

Casts.—There has been some difference of opinion as to the mode of formation of the little cylindrical bodies which are formed in the urine and in the kidney tubules. The question has been whether they are all formed of substances coagulated from the blood plasma, or whether some are formed of substances derived from the renal epithelium. Certainly most of them are formed from the blood plasma. They are composed of a transparent, homogeneous matter with which may be mixed renal epithelium, white and red blood cells, and the granular matter, fat, and nuclei derived from degenerated epithelium. The presence of casts in the urine means, therefore, that there has been an exudation of blood serum into the kidney tubules and more or less degeneration of the renal epithelium. The number of casts in the urine is usually an indication of the number formed in the kidneys, but not always; we may find but few casts in the urine during life, and yet after death the kidneys are seen to contain a large number. Albumen and casts are usually present in proportionate quantities: if there is much albumen there are generally many casts, but albumen may be present in large quantities with very few casts. The centrifugal machines which are now in use are of great assistance in looking for casts.

Any one who wishes to understand casts and their mode of formation must look at them in kidney sections, as well as in the urine. It seems hardly necessary to warn against confounding cylindrical strings of mucus formed in the bladder, often having crystals imbedded in them, with casts formed in the kidney tubules, but the mistake is sometimes made.

Hyaline casts in small numbers, like albumen in small quantities, are occasionally present without disease of the kidneys.

Acute congestion of the kidneys often gives hyaline casts, sometimes granular and nucleated casts.

Chronic congestion gives a few hyaline casts.

Acute degeneration gives casts according to its severity—hyaline casts only, or granular and nucleated casts, or epithelial and blood casts.

Chronic degeneration gives only a few hyaline casts, or none at all.

Acute exudative and acute diffuse nephritis give many casts of every kind.

Chronic nephritis with exudation gives many casts of all kinds, their number much increased when there is an exacerbation of the nephritis.

Chronic nephritis without exudation gives a few hyaline casts or none at all.

Dropsy.

The association of dropsy with kidney disease is of such frequent occurrence that it is often difficult to convince both patients and physicians that ''Bright's disease'' can exist when dropsy is absent.

If we go through the list of diseases of the kidney we find that their association with dropsy is as follows:

Acute congestion of the kidney—no dropsy.

Chronic congestion of the kidney—dropsy according to the condition of the heart.

Acute and chronic degeneration of the kidney—no dropsy.

Acute exudative nephritis—subcutaneous dropsy, most frequent with nephritis caused by scarlet fever, or by exposure to cold. A similar subcutaneous œdema can be produced by scarlet fever or by exposure to cold without nephritis.

Acute productive (or diffuse) nephritis—both subcutaneous dropsy and dropsy of the serous cavities.

Chronic nephritis with exudation—dropsy of the subcutaneous connective tissue and of the serous cavities in the majority of cases.

Chronic nephritis without exudation—no dropsy until late in the disease, unless from complicating lesions.

Suppression of urine from obstruction of the ureters—no dropsy.

The primitive explanation of renal dropsy was that fluids accumulated in the body because the patient passed too little water. This explanation has always had its effect on therapeutics; the rule has been, if a patient has dropsy make him pass more urine. But the matter is not as simple as this.

The present condition of our knowledge on this subject may be stated somewhat as follows:

1. All dropsies are due to an increased transudation of blood serum from the capillaries and a diminished absorption by the lymphatics. The increased transudation is the more important part of the process.

2. Inflammatory dropsies (or exudations) and passive dropsies may be produced in one or two ways:

(*a*) The capillaries act as filters. Either increased pressure, a change in the composition of the serum, or a change in the walls of the capillaries can cause an increased transudation through the walls of the capillaries.

(*b*) The capillaries with their endothelium act as glands and secrete serum. Changes in the composition of the blood or irritating substances in the surrounding tissues can irritate the endothelium and cause increased secretion.

Following these rules, the probable explanations of renal dropsies are:

1. In acute exudative nephritis the dropsy is regularly confined to the subcutaneous connective tissue. It is not always present. It is due to inflammatory changes in the skin, to irritating substances in the blood, or to changes in the walls of the capillaries.

2. In acute productive nephritis and in chronic nephritis with exudation the dropsy involves both the subcutaneous connective tissue and the serous cavities. It may be due to irritating substances in the blood, to anæmia, to changes in blood pressure, or to changes in the walls of the capillaries.

3. In chronic nephritis without exudation the dropsy comes on,

if at all, late in the disease, and is due to changes in the blood pressure.

The rule is that whenever there is much general dropsy there is also a large quantity of albumen in the urine. In other words the same causes, which effect transudation of serum from the blood vessels in other parts of the body, act also on the renal vessels and produce a transudation of serum which is mixed with the urine. ✓

Scattered through medical literature are reports of cases of general subcutaneous dropsy coming on suddenly, lasting for a short time, without any evidence of renal or other disease, and terminating in recovery. Traube thinks that such dropsies are due to a disturbance of the function of the skin caused by exposure to the weather, but this explanation will not answer for all the cases. In some patients (Taylor, *Medical Times* and *Gazette*, 1871) the dropsy was preceded by a well-marked febrile movement. I only know of one case of this kind which terminated fatally. It is reported by Wernicke (*Deutsch. Archiv fur klinische Medicin*, VI., 622). The patient, a woman twenty-two years old, died apparently from the dropsy, and the autopsy showed no lesion to account for the symptoms. The following case is an example of this form of dropsy:

CASE I.—Male, aged 26, admitted to the Roosevelt Hospital on April 17, 1884. Had been perfectly well and working hard two months ago. Then he began to have a cough, mucous sputa, and wheezy breathing. One month ago he developed general subcutaneous œdema; the urine was somewhat diminished in quantity, but the man did not feel sick. When admitted to the hospital his pulse was 96, temperature 98° F., respiration 30. There was very marked general subcutaneous œdema. The skin and mucous membranes were rather pale, but the man was well-nourished and did not feel at all sick. A soft systolic murmur could be heard at the apex and base of the heart; the action of the heart was somewhat intermittent and irregular. The dropsy increased for a few days and then gradually diminished. The record of the urine was as follows:

Date.	Quantity in Ounces.	Albumin.	Specific Gravity.	Urea in Grains.
April 19	68	None	1.024	608
" 20	42	Trace	1.012	...
" 21	96	"	1.022	340
" 22	150	"	1.010	351
" 23	130	"	1.026	280
" 24	84	"	1.008	479
" 25	132	None	1.014	672
" 26	136	"	1.012	858
" 27	62	"	1.014	410
" 28	44	Trace	1.016	394
" 29	45	None	1.014	...
" 30	...	"	1.016	...

By May 1 the dropsy had entirely disappeared and the man was apparently well.

I have seen a number of hospital patients who unquestionably had kidney disease, but who had attacks of subcutaneous œdema after exposure, apparently not connected with their kidney disease, but caused by inflammation of the skin.

The ordinary treatment of dropsy is directed to the removal of the serum after it has transuded from the vessels. We try to get rid of the dropsy by sweating, by diuresis, or by purging. It is evident that a much more satisfactory treatment would be to prevent the transudation. If we could find remedies to destroy the irritating substances in the blood and tissues which cause the blood serum to transude, we would be able to prevent the dropsy instead of having to get rid of it.

ALBUMINURIA.

Since the time of Richard Bright the presence of albumen in the urine has been regarded as a proof of kidney disease both by physicians and by the laity; and in spite of all evidence to the contrary this is still the popular belief. It is true that any educated physician will now admit that albumen may be absent with kidney disease and present without it, but this admission is largely theoretical. In practice the old belief makes itself felt, and the presence of albumen is still looked for as the main evidence of disease of the kidneys.

The general belief concerning the albumen has been that it is removed from the blood by the kidneys just as urea or sugar is, and if large quantities of it are removed from the blood the composition of this fluid is changed. A good deal of pains has been taken to discover why it is that diseased kidneys should excrete albumen.

As a matter of fact, the presence of serum albumen and serum globulin in the urine means that the blood serum, of which they are constituents, has become mixed with the urine. The simplest way in which this can happen is to have bleeding from the bladder or kidneys so that the blood and urine are mixed. The ordinary way is for the blood serum to transude from the kidney capillaries just as it does from capillaries in other parts of the body. Albumen in the urine, therefore, means the same thing as serum in a serous cavity—that there has been a transudation of serum from the vessels. To keep the matter clear in our minds, whenever we use the word albuminuria we should do so with the idea that it is a popular way of saying that the urine has blood serum mixed with it. In this way we will think of the kidney as we do of other parts of the body—as liable to exudations of serum either of inflammatory or of dropsical character. But, just as in the serous membranes the exudation is not in the membrane but in its cavity, so in the kidney the exudation is not into the parenchyma but into the tubules.

THE CAUSES of albuminuria, therefore, are the same as the causes of dropsies in all parts of the body:

1. *Changes produced by inflammation in the walls of the capillaries* which render them more permeable. In this way are produced the albuminuria of both forms of acute nephritis, of the severe forms of acute degeneration, of acute congestion, and of some of the cases of acute and chronic nephritis with exudation.

2. *Changes in the composition of the blood,* causing either increased filtration or increased secretion of serum. This would account for the albuminuria of anæmia, of puerperal eclampsia

without nephritis, of the mild cases of acute degeneration, of some of the cases of acute and chronic nephritis with exudation.

3. *Changes in blood pressure.*—This would be the probable cause of the albuminuria in some of the puerperal cases, in chronic congestion, in some of the cases of chronic nephritis with exudation, and in chronic nephritis without exudation.

4. *Non-inflammatory changes in the walls of the capillaries,* rendering them more permeable to the escape of serum. Such changes would account for the albuminuria which is found without inflammation of the kidneys, changes in the blood, or alterations in the blood pressure.

The significance of albumen in the urine depends, therefore, altogether upon its causation. As a symptom it may be compared to cough. It is well known that, while cough is a frequent symptom of disease of the lungs, yet its presence does not tell us what the disease of the lungs is, nor does it even tell us that there is necessarily disease of the lungs at all. Albuminuria, while it always means that the capillaries of the kidneys allow the blood serum to transude through their wall, does not tell us the cause of this transudation. The study of this causation is practically a study of the causes of dropsy.

Albuminuria Without Disease of the Kidney.

The examination of the urine by the physicians of life insurance companies, and by other physicians who have examined this excretion in cases of school children, of soldiers, and of other groups of persons, has brought out the fact that albumen is present in the urine in many persons who have no disease of the kidneys. These persons can be arranged in the following groups:

1. *Paroxysmal or Cyclic Albuminuria.*—The characteristic features of this form of albuminuria are: that the quantity of albumen is large, while casts may be present or absent; that if we examine the urine at regular intervals during the twenty-four hours there is a regular rise and fall in the quantity of albumen. The albumen begins to appear soon after rising in the morning,

increases through the day, falls after going to bed, disappears at night, and reappears again the next morning. This regular cycle can be disturbed by changing the hours of rest, of meals, and of exercise. The rule is that the appearance of the albumen is favored by exercise and by eating, while rest in bed causes it to disappear. There seems to be no way of accounting for this form of albuminuria, except by supposing that there are changes in the composition of the blood, or in the walls of the renal capillaries.

The persons in whom this form of albuminuria is present are regularly young males, who also suffer from more or less disturbance of the general health. The patients suffer from anæmia, lose flesh and strength, have headaches, neuralgic pains, bodily and mental languor, hysteria, and disturbances of the functions of the stomach, liver, and intestines. But there is a great difference in the patients as to how far these additional symptoms are developed. In some they are but trifling, in others they are well marked.

To distinguish these patients from those who have a true nephritis is by no means easy; the diagnosis may remain doubtful for months, and even then it is difficult not to make mistakes.

The treatment of these patients consists in the regulation of the diet and mode of life, and the management of the disturbances of digestion and of the condition of the blood. The diet should be liberal and varied, but all indigestible articles of food must be excluded. Massage, hot and cold baths, and regulated exercise are to be systematically carried out. A climate which admits of many hours' daily exposure to the open air and sunlight is to be preferred.

All disorders of digestion are to be remedied as far as possible.

The change in the composition of the blood is not marked; neither the quantity of hæmoglobin nor the number of blood cells is much diminished; iron is of service in the treatment of the affection, but does not act as a specific.

The prognosis as regards the life and health of these patients is good, but it may be very hard to get rid of the albumen altogether.

Case II.—Male, 19 years old, April 18, 1902. Three years ago, when apparently in good health, he was refused by a life insurance company on account of kidney disease. He was put under treatment for this with arsenic and other drugs and his diet was restricted. Under this management he lost flesh and strength, but had no dropsy or other renal symptoms.

The urine was examined daily for about a month. The specific gravity was always close to 1.025, the urea was not decreased, every morning there was a trace of albumen and a few casts, every evening there was a considerable quantity of albumen and a good many casts.

He was told to take three good meals a day, no medicine, and to live out of doors.

December 4, 1902: His general health is very good and he is working hard downtown. There is still a little albumen in the evening urine.

October 4, 1903: His health continues to be good, and there is a little albumen in the evening urine.

2. *Dietic Albuminuria.*—This occurs both in children and in adults. It may follow the ingestion of only certain kinds of food—cheese, pastry, and eggs; or of any kind of food; or of any food which is not properly digested; or it may occur when exercise follows immediately upon the ingestion of food. The quantity of albumen is small and there are few or no casts.

If this form of albuminuria is temporary, it is not a serious condition, but if the disposition to it persists, the patients are to be regarded with suspicion. They are also liable to temporary glycosuria; they may have well marked functional disturbances of the liver; they may have the gouty disposition; or they may have cirrhosis of the liver, or chronic arteritis.

The treatment consists in regulating the diet and exercise in the same way as in persons with the gouty disposition; in relieving constipation; and in the use of the drugs which are likely to increase the production of bile.

3. *Albuminuria after Exertion.*—The exertion must be severe and prolonged, in long and fatiguing marches by soldiers; prolonged contests in walking or running; violent exercises, such as boxing or wrestling. The quantity of albumen may be considerable and numerous casts may also be present. It seems prob-

able that this form of albuminuria is due to a congestion of the kidneys caused by the exertion. After the cessation of the exertion the albumen regularly disappears within a few hours or days.

4. In a considerable number of the cases of simple anæmia of young women albumen and casts are found in the urine. As the anæmia is cured by the administration of iron the albumen and casts disappear.

5. *The Albuminuria of Adolescents.*—It has been recognized by many observers that an albuminuria lasting for months is of very frequent occurrence in boys and girls, without disease of the kidneys and without impairment of the general health.

There can be no question that these children do best with plenty of good food and out-of-door exercise. There may be a little anæmia, but it does well without the use of iron.

6. In a considerable number of adults small quantities of albumen and a few casts are found in the urine from time to time without any other evidences of kidney disease. These patients need watching, but I have followed some of them for many years and have seen no evidence of kidney disease. These patients should give up any bad habits they may have, but otherwise they can go on with their ordinary mode of life.

CASE III. *Albuminuria, with good health for twenty-seven years.*—Male, 23 years old, physician, came to me on November 26, 1880.

Four years before that time, while feeling perfectly well, he happened to test his urine and found that it contained a considerable quantity of albumen. Since that time albumen has been constantly present, but no casts. He has continued to feel well and to take a good deal of exercise.

In November, 1880, the urine was rather scanty, 16 to 24 ounces, specific gravity 1.030 to 1.035, a good deal of albumen but no casts. He is somewhat anæmic. Under the use of iron the anæmia improved and the albumen diminished. Now, in December, 1903, his health has been, and is, perfectly good. There is no enlargement of the heart and no thickening of the arteries. The urine still continues to contain appreciable quantities of albumen and a very few hyaline casts. It contains ten grains of urea to the ounce.

URÆMIA.

It is well established that the principal function of the kidneys is to remove from the body a quantity of excrementitious substances. It is equally well established that a number of the diseases of the kidney interfere with this function and allow the excrementitious substances to accumulate in the blood and tissues. It is a matter of daily observation that persons who suffer from kidney disease exhibit symptoms of such a character as to give the idea that these persons are in some way poisoned. The sequence seems to be logical: disease of the kidneys, failure to eliminate excrementitious substances, accumulation of such substances in the blood and tissues, poisoning of the body by these substances, the development of symptoms due to the poisoning. To such morbid process the name of "uræmia" can properly be given. So, in the year 1894, we find the following definition of uræmia in Dunglison's Medical Dictionary: "Certain morbid phenomena, implicating the nervous centres more especially, due to retention of excrementitious substances in the blood which are normally excreted by the kidneys, as in Bright's disease." And this definition fairly represents the popular belief concerning uræmia.

Unfortunately it is not possible to dismiss the subject in this easy way. We are confronted with many contradictions difficult of explanation, and a review of the history of the subject shows that these difficulties have always been felt.

The simplest explanation of the phenomena of uræmia—that they are due to the presence of urea in the blood—has been contended for by many observers from the time of Christison down to the present moment. The proof has been derived from the examination of the blood in human beings, and from experiments on animals.

It has been demonstrated over and over again that the blood of persons suffering from uræmic attacks may contain a large excess of urea, that their serous effusions may contain large quantities of urea, and even that the urea may appear as a dry powder on the surface of the skin. It has also been shown that in a number of cases the outbreak of uræmic convulsions is preceded by a diminution in the excretion of urine and of urea.

The experiments on animals have consisted in injections of urea into the veins, in the introduction of urea into the stomach, and in abolishing the function of the kidneys by ligating the blood vessels or the ureters.

The introduction of urea in considerable quantities into the veins or into the stomach is well borne by animals, provided that the kidneys perform their functions. The urea is eliminated with the urine. If, on the other hand, after the injection of urea into the blood the animal is entirely deprived of fluids, or the functions of the kidneys are arrested by operation, then vomiting, diarrhœa, muscular contractions, and death regularly follow.

Ligature of the blood vessels of the kidneys, or of their ureters, or extirpation of the kidneys is followed by an accumulation of urea in the blood and tissues. The animals have vomiting and diarrhœa, become stupid, and die.

The contradictions to this theory of uræmia were soon noted. Owen Rees was one of the first to call attention to the fact that prolonged anuria is not necessarily accompanied with renal symptoms. His illustrative case was a patient in whom one kidney was absent; the ureter of the other kidney became blocked by a calculus, and there was complete suppression of urine. The patient died, but there were no uræmic symptoms. Cases of suppression of urine lasting for a number of days are not infrequent, and the ordinary experience has been that it is precisely in these cases that uræmic symptoms are absent, although death regularly follows.

More than this, Bartels and others have found that the blood drawn immediately after a uræmic attack may contain no excess of urea. It is a matter of ordinary experience that uræmic symptoms may come on in persons who are passing a normal quantity of urine of good specific gravity. So there seems to be no escape from the facts that complete suppression of urine is not regularly followed by uræmic symptoms, and that uræmic symptoms may occur without an excess of urea in the blood, or a diminution in the excretion of normal urine.

So far as the experiments on animals are concerned, they seem merely to show that urea in the blood does but little harm, and that abolition of the functions of the kidneys causes death.

It was to escape from some of these difficulties that Frerichs proposed the explanation that the cause of uræmic symptoms was poisoning by carbonate of ammonia. He taught that urea in excess in the blood did no special harm, but that if by the action of some ferment the urea was changed into carbonate of ammonia, then symptoms of intoxication would regularly follow. This theory, at one time popular, is now so entirely abandoned that it is not necessary to state the objections to it.

A modification of the theory of intoxication by urea is that of intoxication by urea and the other excrementitious substances of the urine together. The same affirmative and negative facts are to be found for this theory as for that of poisoning by urea alone. Normal uncontaminated urine injected into the veins of animals seems to do little harm unless the kidneys of these animals are injured by operation. If the kidneys are operated on, the animals die. The urine of both animals and human beings is, however, sometimes poisonous.

In human beings there are the patients with uræmic symptoms and an excess of excrementitious substances in their blood and tissues; the patients with uræmic symptoms, but without any excess of excrementitious substances; and the patients with anuria, an excess of excrementitious substances, and no uræmic symptoms.

A very important modification of the chemical aspect of the question is that made by Oppler and others. They hold that it is an error to think that urea or any other constituent of the urine acts as a blood poison. Rather an interference with the functions of the kidneys must lead to a disturbance of the regular chemical changes in all parts of the body. Such an interference is followed by a change in the nutrition of the tissues which shows itself in loss of weight, in anæmia, and in disturbances of the functions of the brain. This way of looking at the subject is certainly a very rational one.

The opposition to all the chemical explanations of uræmia looks to changes in the blood pressure as the exciting cause of uræmic attacks. The most complete theory of this kind is that of Traube.

This theory explains the occurrence of uræmic attacks as follows: The disease of the kidneys causes thinning of the blood serum. If by any accidental circumstance the blood tension is suddenly increased, or the blood serum still further thinned, œdema and anæmia of the brain are produced. The form of the uræmic attack will vary with the portion of the brain which is rendered anæmic or œdematous. If the cerebral hemispheres alone are involved the patient simply becomes comatose; if the central portions of the brain alone are affected there will be convulsions without coma; if both the hemispheres and the central portions of the brain are anæmic and œdematous, both convulsions and coma are developed.

Traube also states:

That he never saw an attack of uræmia in renal disease where the left ventricle of the heart was not hypertrophied, and where an increase of tension in the aortic system could not be demonstrated.

That the diluted state of the blood serum can be recognized by the pallor of the skin and mucous membranes and the presence of dropsical effusions.

That in every instance in which he examined the brain after death he could confirm the existence of anæmia and œdema.

That the presence of blood effusion within the cranial cavity in many of the cases confirms the suspicion that the abnormally high arterial blood pressure to which these effusions owe their origin have also something to do with the production of the œdema which is present at the same time.

Experiments on animals have also shown that by ligating the ureters, then the jugular vein on one side, and then injecting water into the carotid on the same side, general convulsions and coma can be produced. After death œdema of the brain without extravasation of blood is found.

The objections to Traube's theory are obvious: In patients who exhibit well-marked cerebral symptoms the specific gravity of the blood serum is not always lowered; the arteral tension is not always increased; neither anæmia nor œdema of the brain can always be demonstrated after death. These are facts which are soon ascertained by any one who sees much of kidney disease.

The marked uræmic symptoms which occur at the close of pregnancy, and are known under the name of puerperal eclampsia, differ from ordinary uræmic attacks in that they may occur without marked structural changes in the kidneys. A variety of explanations have been offered as to their causation.

Some very good experimental work on uræmia has been done by J. R. Bradford (*Lancet*, March, 1898). He has found that excision of a portion of one kidney is followed by a slight increase of the urinary water, without increased excretion of urea, or a deterioration of the health of the animal. Excision of portions of both kidneys is followed by great and permanent increase in the quantity of urinary water without any increased excretion of urea or change in the general health. The urine of these animals is like the urine of insipid diabetes or that of some of the human kidneys, in which chronic nephritis has caused decided atrophy of the kidneys. There is a slight increase in the quantity of urea in the blood. If, however, the excision is so large that only one-fourth of the original total of kidney weight is left, then not only is the quantity of urinary water increased, but the quantity of urea excreted is also increased. In these animals an increased quantity of urea is found in the blood and in the tissues. The animals die feeble and emaciated, but without uræmic symptoms.

Complete suppression of urine caused by removal of both kidneys or ligation of both ureters causes a marked increase of urea and of other urine extractives in the blood and tissues. The increase is greater than would be due to the retention alone. The animals die feeble, emaciated and with a low temperature.

He found that in normal human blood the percentage of urea is 0.015. In five cases of suppression of urine due to calculi and similar causes the percentage of urea in the blood rose from 0.15 to 0.44.

In one case of acute Bright's disease with suppression of urine, the urea in the blood was 0.35 per cent.

In cases of chronic Bright's disease at times when the patients were having no symptoms and apparently in good health the quantity of urea in the blood was somewhat increased.

In twenty-five cases of fatal uræmia without dropsy he found the quantity of urea in the blood to be from 0.2 to 0.5 per cent.

His final conclusions are: that uræmia cannot be held to be due to simple retention of urea and other urine extractives; that in kidney diseases and after experimental lesions of the kidney there is evidence of great tissue disintegration, and that it is more than probable that uræmia is dependent upon this directly or indirectly.

There is a practical advantage in separating the cases of uræmia into two classes—those of acute uræmia, and those of chronic uræmia.

In acute uræmia the symptoms come on in attacks of greater or less severity, and of longer or shorter duration, and are accompanied by one or more of the following symptoms:

1. *Headache and sleeplessness*, which come on in attacks lasting for days or weeks. The headache may be of mild type, or very severe. In extreme cases the pain is so severe and the sleeplessness so distressing that the patients are almost maniacal.

2. *Hemiplegia and Aphasia*. These two symptoms, which may occur separately or together, are seen in patients who have chronic nephritis without exudation, and in women with puerperal eclampsia. The invasion of the hemiplegia is sudden, and it is usually accompanied by coma. There is loss of motion alone, and of both motion and sensation. The hemiplegia, coma,

and aphasia may continue up to the time of the patient's death, or disappear after a few days. In the latter case the patient may have several such attacks. In chronic nephritis the hemiplegia may occur either early or late in the course of the disease. In these patients chronic endarteritis, especially of the cerebral arteries, is very regularly present. I think that it is probable that the hemiplegia is due to the endarteritis, rather than to the kidney disease.

3. *Sudden Blindness.*—Besides the loss of vision due to nephritis retinitis, there may be a sudden blindness which lasts for hours or days. In these patients no anatomical changes in the eyes have been discovered. This form of blindness is not uncommon in puerperal eclampsia; it occurs in a moderate number of the cases of chronic nephritis. We do not understand the nature or treatment of this blindness; fortunately it only lasts for a short time.

4. *General Epileptiform Convulsions.*—These have always attracted much attention as one of the most terrible and dangerous of the results of kidney disease. They may accompany either form of acute and chronic nephritis and pregnancy.

5. *Contractions of Groups of Muscles.*—These are of common occurrence in the severe forms of acute and chronic nephritis and in puerperal eclampsia. Very often they merely precede an attack of general convulsions. They are best marked and of longest continuance in the advanced cases of chronic nephritis with exudation.

6. *Delirium and Coma.*—They come on suddenly in attacks, either associated with convulsions or by themselves; or they are developed slowly and gradually and continue for a considerable length of time. The attacks belong to the severe cases of acute nephritis, to the exacerbations of chronic nephritis with exudation, and to the ordinary cases of chronic nephritis without exudation. The gradual development of delirium and coma is apt to continue, although sometimes with intervals of improvement up to the time of the patient's death. They are very often seen in the advanced stages of both forms of chronic nephritis.

7. *Vomiting* is seen in many cases of nephritis. It is evidently

due to a number of different causes. In acute nephritis the vomiting seems to be of the same character as that which may occur with an acute inflammation of any part of the body. In chronic nephritis the vomiting may be due to chronic gastritis; or the stomach, like the intestine, gets rid of some of the accumulated urea and serum.

Besides these forms of vomiting there is a special and aggravated form which belongs regularly to the cases of chronic nephritis without exudation. It is usually accompanied by a marked increase of arterial tension. The vomiting is frequent, distressing and may continue for days. The most efficient means of controlling it are chloral hydrate by the rectum in twenty-grain doses, or hypodermic injections of morphine.

8. *A Rise of Temperature.*—In acute nephritis, as in any other acute inflammation, there may be a febrile movement. As a rule the temperature is not high, and falls to the normal within a week. But in children temperatures of 104° or 105° F. may be reached.

In chronic nephritis the severe attacks of cerebral symptoms —headache, convulsions, delirium, coma, hemiplegia—are not infrequently accompanied with a considerable rise of temperature. I have seen as high as 109° F., and yet the autopsies show no reason for the febrile movement.

9. *Dyspnœa* is one of the most frequent and interesting of the symptoms of nephritis. There is a dyspnœa due to fluid in the pleural cavities, or to œdema of the lungs; a dyspnœa due to the pressure on the diaphragm by fluid in the abdomen; and a dyspnœa due to bronchitis or to contraction of the bronchi; but the most important form of dyspnœa is one which is independent of all these causes, and is directly caused by disturbances of the circulation. It belongs to both forms of chronic nephritis, but is more common with nephritis without exudation. The patients, as a rule, in addition to the nephritis have pulmonary emphysema, chronic arteritis, hypertrophy of the left ventricle, or chronic endocarditis. Not infrequently one or more of these lesions is much more advanced and apparently more important than the kidney disease. Especially is this the case with chronic

arteritis, which gives many of the most marked examples of this form of dyspnœa with but very little nephritis.

The dyspnœa may come on at any time in the course of a nephritis; very often it is the first symptom which causes the patient to seek medical advice. It begins with attacks, which at first are brought on by bodily and mental exertion, or come on of themselves at an early hour in the morning. In none of the attacks do we hear the characteristic breathing of bronchial asthma. The attacks at first only last for an hour or so, and during the rest of the day the breathing is comfortable. But even in these mild attacks the patient cannot lie down. As time goes on the attacks become more frequent and of longer duration. Finally comes the terrible period when the breathing is always bad, the patients cannot lie down at all, and yet go on living for weeks and months.

Increased Arterial Tension. This is not always classed with the uræmic symptoms. It is, however, one of the most frequent and important of the symptoms of chronic nephritis; and it is by it that many of the so-called uræmic symptoms are produced.

It has been believed that this increased tension of the blood in the arteries is due to chronic changes in the walls of the arteries and capillaries which interfere with the passage of the blood through them. The explanation is very probably true up to a certain point, but it does not account for the attacks of increased arterial tension which come and go within a few hours. I do not see how these can be produced except by the temporary contraction of arteries which have a well-developed muscular coat, such as the radial artery. I think that it is possible to demonstrate after death in such arteries an hypertrophy of the muscular coat, in patients who have had many attacks of increased arterial tension.

If this is admitted, then we have to find a reason for the attacks of contraction of the arteries which last for hours or for weeks, and which can often be controlled by the drugs which dilate the arteries.

Such attacks of contraction of the arteries occur with:

Attacks of angina pectoris.

Chronic endocarditis.
Chronic arteritis.
Pulmonary emphysema.
Chronic nephritis.

The fact that the symptoms occur in attacks which are often temporary; that they occur without relationship to the quantity of urine or of urea excreted, in persons who have very little kidney disease, and even in those who have no kidney disease, seems to point to the existence of a poison which is not due to a nephritis but to some other cause. It seems probable that the same poison may be the cause of both the uræmic symptoms and the nephritis, and that it may cause the uræmic symptoms before it causes the nephritis. We see often enough cases like the following, which it is difficult to account for in any other way. A middle-aged man for some months suffers from attacks of bronchial asthma, without bronchitis; from rapid heart action without hypertrophy or murmur; from large quantities of albumen, with urine of a specific gravity of 1.025, 50 ounces in the twenty-four hours, and ten grains of urea to the ounce. After only a week's treatment the patient has no more asthma, a normal heart action and a trace of albumen in the urine. Or we may see a patient with contracted arteries, a laboring heart and dyspnœa, who passes urine containing 2 per cent, of urea and without albumen or casts.

The poison of acute uræmia seems to produce the symptoms directly by acting on the brain, and indirectly by causing contraction of the arteries so that the whole condition is a complex one. We have no antidote for the poison, but we know by experience that many of the attacks of acute uræmia can be controlled and terminated by the use of drugs which dilate the arteries.

Chronic uræmia comes on, not in attacks, but slowly and gradually. When the condition is once developed it regularly continues until the death of the patient. Headache, sleeplessness, delirium, stupor, coma, muscular twitchings, general convulsions, a temperature rather below than above the normal, arteries which are not contracted, and a feeble heart make up the clinical picture. The patients excrete much less than the normal quantity of urea for each twenty-four hours. The symptoms seem to be caused by the direct action of the poison on the nervous centres without contraction of the arteries. It is a condition over which treatment has very little effect. It would seem, therefore, that in chronic uræmia the symptoms are caused by the failure of the kidneys to eliminate urea and other excrementitious substances from the blood, these substances acting as poisons. In acute uræmia, on the other hand, the kidneys do excrete these excrementitious substances and the poison which causes the symptoms is as yet unknown to us. Another interesting point of difference between these two forms of uræmia is that patients with chronic uræmia are easily poisoned with small doses of opium, while patients with acute uræmia bear large doses with impunity.

CLASSIFICATION OF DISEASES OF THE KIDNEYS.

It is impossible to describe the diseases of the kidneys without some sort of classification. At the present time, however, there is no general agreement as to what this classification should be, nor can such an agreement be reached until our knowledge is more exact than it now is.

The best that we can do is to follow some scheme, which is convenient for clinical purposes, is not at variance with pathological anatomy, and does not present too many exceptions to general rules.

acute

As regards acute nephritis, it seems evident that it is caused by poisons which circulate in the blood. These poisons are: inorganic—arsenic, mercury, phosphorus, etc.; organic—the toxins of the infectious diseases, the poisons of pregnancy and the unknown poisons of the primary cases.

These poisons, according to their dose and virulence, produce acute congestion of the blood vessels, degeneration or death of the renal epithelium, exudation from the blood vessels, changes in the Malpighian bodies, or a new growth of connective tissue in the stroma. It is to be remembered that each poison may produce any one of these changes, although it often has a preference for one rather than another; and that, on the other hand, the same anatomical changes may be produced by several of the different poisons. The toxin of scarlet fever may cause changes in the epithelium alone, in the epithelium and blood vessels alone, or in the epithelium, blood vessels and stroma. Changes in the epithelium, blood vessels and stroma may be caused by the toxins of pregnancy, or of diphtheria.

We divide the cases of acute nephritis, therefore, into cases of:

Acute Congestion.
Acute Degeneration.
Acute Exudative Nephritis.
Acute Productive Nephritis.

We hold that these do not run into or succeed each other, but are different from the outset, and continue different to the end. We hold also that it is of great practical importance to recognize the toxin which causes the nephritis. For experience teaches us that each toxin has a preference for one of the forms of nephritis rather than another. Pneumonia and typhoid fever regularly produce acute degeneration of mild type; yellow fever and acute yellow atrophy of the liver produce acute degeneration of severe type; scarlet fever causes acute exudative nephritis; pregnancy and the poison of the primary cases cause acute productive nephritis.

As regards the cases of chronic nephritis we find that a certain number succeed an attack of acute productive nephritis after a shorter or longer time; that some are caused by prolonged interference with the circulation of the blood; and that some are due to the same causes which produce waxy degeneration of the viscera. Besides these every one admits that there are at least two great classes of chronic nephritis: one in which the kidney is more or less large and white, while during life there is dropsy and much albumen in the urine; and one in which the kidney is more or less contracted and red, with little or no albumen in the urine and little or no dropsy. It has become customary to use the names of large white kidney or parenchymatous nephritis for one of these forms of nephritis; and of granular or atrophied kidney, or interstitial nephritis for the other.

Any one who looks at a sufficient number of these kidneys will find that there are a small number in which only the epithelium is changed, but that with these albumen and dropsy are not often associated; that in all the others the epithelium, Malpighian bodies and stroma show marked changes; that the size, color and gross appearance of these kidneys are in no certain relation to the clinical symptoms. Thus Bamberger, from a study of 2,430 cases of chronic nephritis, exclusive of cases of chronic congestion and amyloid degeneration, concludes that: in all the large, smooth kidneys with thickened cortex there is a growth of interstitial connective tissue, atrophy of the glomeruli and thickening of the arteries; that there are cases with the history of atrophied kidney, but at the autopsy large white kidneys, or waxy kidneys are found; that there are cases with the history of large white kidneys, but at the autopsy the kidneys are found to be atrophied; that there are cases with mixed histories of both large white and atrophied kidneys.

On the other hand, one cannot practice medicine without seeing that there are really two different forms of chronic nephritis; which are not related to each other, nor are they modifications of each other.

It seems to me that we get the effect that all the cases of chronic nephritis are caused by two poisons, or two sets of poisons.

One of these poisons, or sets of poisons, has as its marked characteristic the property of causing exudation from the blood vessels —albuminuria and dropsy. This property is most marked if the poison acts quickly; it is less evident if the poison acts slowly. So in this form of nephritis while albuminuria and dropsy are the rule, they may be absent while the disease is going on very slowly.

We do not know in the least what this poison or poisons are, so we call the condition a "chronic nephritis with exudation."

The other poison, or set of poisons, seems to be of the same nature as that which causes chronic productive inflammations in other parts of the body—endocarditis, arteritis, cirrhosis of the liver, etc. Here again we do not know the exact nature of the poison, so we call the nephritis a "chronic nephritis without exudation."

It must also, I think, be admitted that some of the symptoms of chronic nephritis are due, not to the nephritis, but to the poisons which cause the nephritis.

Whether we can go farther than this and say that there is such a thing as Bright's disease, with which disease of the kidney may or may not be associated, is still a matter open to discussion.

There is a group of persons who are more liable than are others to chronic productive inflammation in different parts of the body. It may be the lungs, or the heart, or the arteries, or the liver, or the kidneys that are affected; and either one or several of these organs are involved at the same time.

The liability is most common after forty-five years of age, but is by no means infrequent in younger persons. Unquestionably many of these persons are gouty; in some there is a history of chronic alcoholism; in some there is an hereditary history; many of them suffer from disturbances of digestion; many of them have habitually an excess of urates or oxalates, or occasional sugar or albumen in the urine.

So great is the number of these cases, and so constantly are these persons under observation, that it is often not difficult to recognize that an individual belongs to this group before he has developed any one of the characteristic inflammations. We can predict beforehand that a given individual will, at some time, develop emphysema, or chronic endocarditis, or endarteritis, or cirrhosis of the liver, or chronic nephritis.

It is possible that this group of persons are really all suffering from the same disease, and that they develop the characteristic lesions as the result of the disease. According to this view a person can have Bright's disease while the kidneys are still normal.

There can be no question that this group of cases is a very important one, and it is quite true that most of them do have disease of the kidneys before they die.

There is a well-marked disposition on the part of some authors to include cases of chronic inflammation of the arteries under the name of Bright's disease. This view of the matter is clearly stated by Mahomed in "Guy's Hospital Reports for 1880."

He says: "The object of this paper is to prove that in the earlier stages, and in most cases even to their final stage, the urine of what is generally known as chronic Bright's disease with red granular kidney is most commonly perfectly normal. More than this, its object is to prove, either that chronic Bright's disease is not a renal disease, although it frequently gives rise to a renal affection, or else that another disease must be recognized which constantly precedes and prepares the way for Bright's disease, which may be called arterio-capillary fibrosis, or any other name that may be preferred to it."

The cases which Mahomed narrates in his paper seem to be cases of chronic arteritis, with more or less complicating nephritis.

We will, therefore, describe the diseases of the kidneys under the names of:

1 Acute congestion of the kidney.
2 Acute degeneration of the kidney.
3 Acute exudative nephritis.
4 Acute productive nephritis.
C 1 Chronic congestion of the kidney.
C 2 Chronic degeneration of the kidney.
C 3 Chronic nephritis with exudation.
C 4 Chronic nephritis without exudation.

It is not necessary to make a separate class of the waxy kidneys. For clinical purposes they can all be included with the two forms of chronic nephritis.

1 ACUTE CONGESTION OF THE KIDNEYS.

Definition.—A temporary congestion of the blood vessels of the kidney, which may be so severe as to produce exudation of serum and escape of red blood cells.

Etiology.—Acute congestion is caused by the ingestion of certain poisons, by extirpation of one of the kidneys, by severe injuries inflicted on any part of the body, by surgical operations, especially those on the bladder and urethra, and by over-exertion.

Morbid Anatomy.—It is but seldom that we are able to obtain human kidneys in the state of acute congestion, for the condition is not usually a fatal one. In animals, however, the condition can be produced experimentally by cantharidin. It is found that the kidneys are enlarged, that the veins, capillaries, and Malpighian tufts contain an increased quantity of blood, and that the epithelial cells of the cortex tubes are flattened.

(1) Congestion Caused by the Ingestion of Poisons.

Cantharides given internally, or used in blisters or ointment, is rather a frequent cause of acute congestion of both the kidneys and the bladder.

The urine is diminished in quantity; it is passed frequently, in small quantities, with much pain; or it is retained. It contains albumen, a few casts, and blood. Sometimes large, jelly-like coagula are formed in the bladder.

The patients may have a moderate rise of temperature, pain in the back, abdominal pain, nausea and vomiting, diarrhœa. more or less prostration, delirium, and stupor. The severity of the symptoms depends on the quantity of the drug absorbed. In the bad cases of poisoning the condition of the kidneys is not that of hyperæmia, but of actual inflammation.

When there is only hyperæmia the patients are sick for a few days, and the urine soon returns to its natural condition. Turpentine produces symptoms like those of cantharides.

Treatment.—If the poison has been taken into the stomach, that organ is to be emptied and washed out. Warm baths, or a hot pack, and the use of small doses of opium are of service. Camphor in doses of from 2 to 5 grains every three hours has been recommended in cantharides poisoning.

(2) Congestion Following the Removal of One Kidney.

The urine is scanty or suppressed, and contains albumen and casts. The patients are in a condition of prostration which is very alarming. They may remain in this condition for a few days and then recover, the urine returning after a time to its natural condition; or they become more and more feeble, pass into the typhoid condition with mild delirium, and die.

Treatment.—The patients are to be kept quiet in bed on a fluid diet. They should have one or two long hot packs every day, so as to produce congestion of the skin and profuse perspiration; hot irrigations of the rectum and intra-venous transfusion of saline solution are also indicated.

(3) CONGESTION OF THE KIDNEYS AFTER INJURIES, OR SURGICAL OPERATIONS.

It is well known that any operation on the bladder or urethra, even the passage of a catheter, may be followed by suppression of urine, great prostration, and death within forty-eight hours, and that after death no lesion is found except congestion of the kidneys. These cases are not to be confounded with the cases of septic infection and fever, which may also follow operations on the urethra and bladder.

It is not so well known that surgical operations on any part of the body are occasionally followed by suppression of urine, congestion of the kidneys, and death. We do not know that death in all these cases is caused by the congestion of the kidneys, but there can be no question that congestion of the kidneys is produced in this way.

Treatment.—No satisfactory treatment has yet been found for these patients; in fact, the very short time which intervenes between the operation and death hardly gives time for treatment. Intra-venous transfusion of saline solutions and the splitting of the capsules of the kidneys seem to be indicated.

(4) ACUTE CONGESTION AFTER OVER-EXERTION.

Prolonged marches, violent gymnastic exercises, contests of walking and running prolonged over several days, may be followed by the excretion of such a quantity of albumen and casts in the urine as to indicate a temporary congestion. I do not know of any post-mortem observations which corroborate this belief. There are no other symptoms besides the changes in the urine, and these disappear after a few days' rest.

2. Acute Degeneration of the Kidneys.

Definition.—An acute change in the kidneys, characterized by degeneration or death of the epithelial cells of the tubules, to which may be added an exudation from the blood vessels.

Synonyms.—Acute Bright's disease; Parenchymatous nephritis; Parenchymatous degeneration.

Etiology.—The introduction of certain poisons into the body is regularly followed by changes in the cells of the viscera. The poisons which exert this effect are ordinarily mineral poisons, such as arsenic, mercury, and phosphorus; or the poisons of infectious disease, such as diphtheria, typhoid fever, etc. According to the quantity and virulence of the poison received into the body, there are more or less marked changes produced in the cells of the viscera.

Small doses of such poisons, acting only for a moderate length of time, produce simple swelling of the cells. The cells are swollen, more opaque, more coarsely granular. They are not dead, nor broken down, nor do they contain any new substances; the change in their appearance is due to the swelling of the network which forms a part of every cell. Under these circumstances there are either no changes at all in the blood vessels of the viscera, or a slight congestion, with, perhaps, a little exudation of serum.

Larger doses of such poisons, or more virulent poisons, or a longer duration of the action of a poison, are attended by the deposition in the cell bodies of granules of albuminous matter and globules of fat. At the same time there is a change in the nutrition of the cells, and they are often broken and disintegrated. Under these conditions there may be considerable congestion of the vessels and exudation of serum.

Very large doses of such poisons cause the death of the cells of the viscera, a death which may take the form of coagulation-necrosis or of disintegration and breaking down of the cell. With these changes there will often be an excessive congestion of the vessels and a large exudation of serum.

As the kidneys are excreting organs it is rather natural to think that the substances which cause degeneration of the renal epithelium do so because they are excreted by the kidneys. But, as the same poisons produce similar degeneration in many other parts of the body, it seems more probable that the effect of the poison is produced in the same way that it is in the nerves, the muscles, the liver, and the spleen.

The well-known fact that temporary cutting off of the arterial blood from the kidneys in animals is followed by degeneration or death of the renal epithelium, has led to the idea that degeneration of the kidneys, especially in cholera, is due to ischæmia. This seems possible, but it is a theory not at all applicable to most cases of acute degeneration.

It is curious that so many different poisons should act in the same way. There seems to be nothing in common between the poison of corrosive sublimate and that of yellow fever, and yet the changes in the kidneys are practically the same.

The inorganic poisons, arsenic, etc., act according to the size of the dose taken. A small dose produces only moderate degeneration of the renal epithelium, a large dose causes extensive necrosis with considerable exudation of blood serum.

The toxins of the different infectious diseases vary as to the activity of their effect on the renal epithelium, as to the time in the disease when the degeneration takes place, and as to the frequency with which true nephritis is produced instead of acute degeneration. It is a question of much importance whether the same toxin produces degeneration or nephritis according to its dose, or whether two or more different toxins are necessary. In scarlatina and diphtheria, for example, the rule is that acute degeneration comes in the early days of the disease, acute exudative nephritis in the late days of the disease, and acute productive nephritis just after the close of the disease. Does this mean three different toxins, or that the same toxin varies at different stages of the disease, or that the only difference is in the dose?

For clinical purposes the recognition of the fact that acute degeneration is the ordinary lesion of the infectious diseases is of much practical importance.

Morbid Anatomy.—The gross appearance of the kidney varies with the extent of degeneration. In the ordinary, mild cases, such as accompany pneumonia, the kidney is a little larger, the cortical portion a little thicker and paler. In the severe cases, such as accompany acute yellow atrophy of the liver, the kidney is considerably enlarged and more or less congested.

The changes in the renal epithelial cells are: a simple swelling of the cell body, causing it to look larger and more opaque and to take on irregular shapes; an infiltration of the cell bodies with granules of albuminoid matter and fat; a death of the cells, which may take the form of coagulation necrosis or of a disintegration of the cell bodies; a desquamation of the dead cells so that the tubes are filled with them; a formation of hyaline masses in the cells; a growth of new cells to take the place of the dead epithelium. All these changes are most marked in the convoluted tubes.

In the kidneys with extensive necrosis of the epithelium there may also be congestion of the blood vessels and casts in the tubes.

Symptoms.—With the severe acute degeneration which follows the ingestion of large doses of arsenic, mercury, or one of the other inorganic poisons, the urine is diminished in quantity, or suppressed; it contains albumen, casts, and blood; its specific gravity is unchanged. At first the general symptoms belonging to the poison predominate, but as these subside the patients continue to live and suffer more or less severely from the degeneration of the kidney. They become feeble, pass into the typhoid state, and often die.

The acute degeneration which accompanies the infectious diseases such as scarlatina, diphtheria, typhoid fever, pneumonia, etc., is for the most part of mild type and gives no symptoms except the presence of a little albumen and a few casts in the urine. It is of importance to recognize the frequency with which this affection of the kidneys occurs, the slight injury which it inflicts on the patient, and the completeness with which the kidney lesion disappears after the recovery from the primary disease. Much unnecessary anxiety is often felt by physicians because in a case of pneumonia or some other infectious disease they find albumen and casts in the urine. There are fears not only of death from the primary disease, but of the subsequent development of "Bright's disease." If the albumen and casts are due to degeneration of the kidneys anxiety is needless. If the patients recover their kidneys return to their normal condition.

With yellow fever, with acute yellow atrophy of the liver, and occasionally with the other infectious diseases the degeneration is of intense type, with the death of a large part of the renal epithelium and exudation from the vessels. In such cases the urine is scanty or suppressed. It contains albumen, casts, and blood. The patients are very ill, they may have convulsions, delirium, or stupor; they often die. But it is hard to tell how many of their symptoms are due to the complicating kidney lesion.

CASE IV. *Acute Degeneration of the Kidney; Corrosive Sublimate Poisoning.*—Female, 19 years old. On May 6, 1885, she swallowed a solution of corrosive sublimate containing about twenty-five grains. She at once had pain in the throat and abdomen, soon followed by vomiting and purging.

May 8: She has only passed half an ounce of urine since her admission. This contained albumen, blood and many casts.

May 9: One drachm of urine drawn off by the catheter; it contains albumen and many epithelial casts. She has numerous small, painful, bloody passages from the bowels.

The urine continued to be very scanty; the vomiting and dysenteric passages kept on; she became delirious and died on May 12, six days after taking the poison.

Autopsy.—The heart and lungs are normal.

The liver is of medium size, soft, mottled red and yellow. The spleen is normal.

The mucous membrane of the stomach is congested, hæmorrhagic and coated with mucus. The mucous membrane of the whole length of the small intestine is congested and coated with mucus.

A large part of the inner surface of the colon is coated with fibrin.

There is a little fibrin on the peritoneal surfaces of the stomach and small intestine.

The kidneys are of medium size, smooth; the cortex thick and white; the pyramids red.

In the pyramidal portion the epithelium is well preserved and looks normal; in some of the tubes there is cast matter and in some necrotic epithelium.

In the cortex the epithelium of all the tubes is necrotic and detached, filling up most of the tubes. In some of the convoluted tubes there is a growth of large, flat, new cells on the tube wall.

In the Malpighian bodies the tuft and capsule cells are swollen.

CASE V. *Acute Degeneration of the Kidney; Phosphorous Poisoning.*—Male, 45 years old. On February 1, 1887, he dissolved some phosphorous matches in a glass of water and drank the solution.

On February 2 he came to the hospital with frequent vomiting. He continued to vomit, became jaundiced, and died on February 4.

Autopsy.—The heart is normal.

The lungs are congested and œdematous.

The bronchi are trabeculated and contain muco-pus.

The liver is large and yellow.

The mucous membrane of the œsophagus is brown and necrotic.

The mucous membrane of the stomach is brown and necrotic in large patches.

The kidneys are a little swollen.

The epithelium of the cortex tubes is very much swollen, but not detached.

The epithelial cells are infiltrated with coarse granules and small globules of fat.

CASE VI. *Acute Degeneration of the Kidney; Acute Yellow Atrophy of the Liver.*—Male, 22 years old; house physician in a hospital.

November 25: He began to have nausea and pain in the bowels.

November 26: He had headache, pain in the back, a chill lasting for half an hour, followed by a temperature of 104° and a pulse of 144.

November 27: A chill, followed by a temperature of 106°, pulse 156, headache, vomiting of green fluid, urine scanty, no albumen.

November 28: The vomiting continues, jaundice is beginning.

November 29: Vomiting of coffee-ground matter, temperature normal, decided jaundice, severe pain over liver, delirium, urine contains blood and albumen.

November 30, he died.

Autopsy.—The body is deeply jaundiced.

The heart is normal except for an ecchymosis in the visceral pericardium.

The lower lobes of both lungs are congested.

The liver weighs 2 lbs. 10 oz., it is firm, yellow mottled with red; the gall bladder contains a small quantity of thick bile, the large bile ducts are not obstructed, the liver cells are swollen and filled with coarse granules; some are broken down.

The stomach contains a little blackish fluid.

The spleen is of medium size, not soft. The kidneys are large, the epithelium throughout is swollen and granular, many of the large tubes in the pyramids are packed with blood and granular matter.

CASE VII. *Acute Degeneration of the Kidney; Yellow Fever.*—Male, 60 years old, September 1, 1879. The man had been put as keeper on a ship at quarantine which had had yellow fever on board. The cargo had been discharged and the ship disinfected. The man was sick for three days.

Autopsy, four hours after death.—There is some old thickening of the pia mater of the brain.

The aortic and mitral valves of the heart are moderately thickened.

There are extravasations of blood in the pulmonary pleura and in the lower lobes of both lungs.

The liver is of medium size, mottled red and yellow, the gall bladder is empty, some of the hepatic cells are filled with granules and fat globules, some are disintegrated.

The kidneys are large and smooth. In all the tubes the epithelium is very much swollen and granular and in some disintegrated and detached. In some of the cortex tubes there are large, irregular globular masses of transparent matter.

Treatment.—So far as the degeneration of the epithelium is concerned, we know of no treatment which is likely to affect it favorably. But in the severe cases with congestion of the kidneys and scanty urine, it seems to be good practice to use the hot-air baths or the hot pack.

It is evident that acute degeneration of the kidney can be sharply distinguished from all other forms of kidney disease. It is always produced by the introduction of some poison into the body. It is not accompanied by dropsy, contraction of the arteries, or by other renal symptoms. It is not usually dangerous to life. It is a temporary condition not followed by any chronic kidney disease. If the patient recovers from his poisoning the kidneys return to their normal condition.

3 Acute Exudative Nephritis.

Definition.—An acute inflammation of the kidneys, characterized by congestion, exudation of the blood plasma, emigration of white blood cells, diapedesis of red blood cells, to which may be added changes in the renal epithelium and in the glomeruli.

Synonyms.—Acute Bright's Disease; Parenchymatous Nephritis; Tubal Nephritis; Desquamative Nephritis; Catarrhal Nephritis; Croupous Nephritis; Glomerulo-Nephritis; Interstitial Nephritis.

Etiology.—Acute exudative nephritis is frequently a primary inflammation, occurring either after exposure to cold, or without discoverable cause. It may complicate any one of the infectious inflammations or diseases, but is especially common with scarlet fever. It is one of the forms of nephritis which are caused by pregnancy.

Acute exudative inflammation in any part of the body seems to be caused by local irritation, by the presence of irritating substances in the blood, and by changes in the circulation of the blood and in the inflamed portion of the body. Pathogenic bacteria are sometimes present, sometimes absent in the inflamed tissue.

Unquestionably all the infectious diseases are often complicated with inflammations of different parts of the body. The probable causes of these are the chemical poisons produced by the growth of the pathogenic bacteria belonging to each disease. It seems also that the poison of each disease has a preference for particular portions of the body. In rheumatism the joints and heart are regularly inflamed; in measles, the bronchi; in scarlet fever and diphtheria the throat and kidneys.

As regards the presence of bacteria in the kidneys themselves as exciting causes of inflammation our knowledge is uncertain.

Whether nephritis in puerperal women and after exposure to cold is due to disturbances of circulation or to some poison in the blood is not certain.

There are marked differences in the severity of different cases of nephritis. The exudation of serum is larger in one case, the emigration of white blood cells in another. In one kidney the epithelial cells are contracted, in another they are swollen and degenerated. The glomeruli are much more changed in some kidneys than in others. How closely these differences in the lesions correspond to differences in the causation of the nephritis we do not know.

Morbid Anatomy.—In the mild cases we find no decided lesions in the kidney after death.

In the more severe cases the kidneys are increased in size, their surfaces are smooth, the cortical portion is thick and white, or white mottled with red, or the entire kidney is intensely congested. If the stroma is infiltrated with serum, the kidney is succulent and wet; if the number of pus-cells is very great, there will be little whitish foci in the cortex.

In such kidneys we find the evidence of exudative inflammation in the tubes, the stroma, and the glomeruli, all the changes being most marked in the cortical portion of the kidney.

Epith

The epithelium of the convoluted tubes is often simply flattened. As this same appearance is also found in the chronic congestion of heart disease, it seems probable that this change of the shape of the cells is merely due to the inflammatory congestion.

In other cases, not only is the epithelium flattened, but there is also a real dilatation of the cortex tubes. This dilatation involves groups of tubes, or all the cortex tubes uniformly.

In other cases, the epithelium of the convoluted tubes is swollen, opaque, degenerated, and detached from the tubes.

Tubes

The tubes, whether with flattened epithelium or dilated, may be empty. More frequently, however, they contain coagulated matter in the form of irregular masses and of hyaline cylinders. The irregular masses are found principally in the convoluted tubes; they seem to be formed by a coagulation of substances contained in the exuded blood plasma, and are not to be confounded with the hyaline globules so often found in normal convoluted tubes. The cylinders are more numerous in the straight tubes, but are also found in the convoluted tubes. They also are evidently formed of matter coagulated from the exuded blood plasma, and are identical with the casts found in the urine.

The tubes may also contain red and white blood cells.

In the cases in which there is an excessive emigration of white blood cells, we find these cells in the tubes, in the stroma, or distending the capillary veins. This excessive emigration is not necessarily attended with exudation of the blood serum, and so the urine of these patients may contain no albumen. The white blood cells are not usually found equally diffused through the kidneys, but are collected in foci in the cortex. These foci may be very minute, or may attain a considerable size.

glomeruli

The glomeruli regularly are changed. The cavities of the capsules may contain coagulated matter and white and red blood cells, just as do the tubes. The capsular epithelium may be swollen, sometimes so much as to resemble the tubular epithelium, and this change is most marked in the capsular epithelium near the entrance of the tubes.

The most noticeable change, however, is in the capillary tufts of the glomeruli. These capillaries are normally covered on their outer surface by flat, nucleated cells, so that the tuft is not made up of naked capillaries, but each separate capillary throughout its entire length is covered over with these cells. There are also flat cells which line the inner surfaces of the capillaries, although not uniformly, as in the case in capillaries in other parts of the body. Still, in spite of the presence of all these cells, the outlines of the walls of the capillaries are fairly distinct.

In exudative nephritis the swelling and growth of cells on and in the capillaries change the appearance of the glomeruli. They are larger, more opaque, the outlines of the main divisions of the tufts are visible, but those of the individual capillaries are lost.

It is difficult to tell how much these changes in the glomeruli interfere with the passage of the blood through their capillaries.

In most cases of exudative nephritis the patients recover, and the glomeruli return to their natural condition.

In some examples of exudative nephritis we also find a thickening of the walls of the branches of the renal artery within the kidney. This thickening is principally due to a swelling of the muscle cells in the walls of these vessels.

All these changes in the kidneys are of such a character that they are not likely to be followed by a chronic nephritis. On the contrary, after the patients have recovered, the kidneys return to their normal condition.

The points of importance as regards the lesions of acute exudative nephritis are:

(*a*) In many cases, with much albumen and many casts in the urine, the kidneys show no structural changes. It is evident that congestion, exudation, emigration and diapedesis are the only morbid changes.

(*b*) When the changes described in the tubes and Malpighian bodies do exist they are of a temporary character, so that the kidneys easily return to their normal condition.

(*c*) The popular idea that the kidney tubules in an acute nephritis are filled and blocked up with desquamated epithelium and cast matter is not usually true. Very often the epithelium is flattened and most of the tubes are empty. When the epithelium is swollen and detached very few of the tubes are much filled.

Symptoms.—1. There are cases of acute nephritis of so mild a character that they may easily be overlooked. I think that these mild cases occur more frequently than is commonly supposed. The patients are hardly sick enough to go to bed. They have a little headache, perhaps some aching in the back and limbs, loss of appetite, a little nausea, and the feeling of a general malaise. They often think they have taken cold. These indefinite symptons last for one or two weeks, disappear, and the patient is well again. If the urine is not examined it is not known that the patient has been suffering from a mild nephritis. If the urine is examined it is found that the quantity is somewhat diminished, the specific gravity is not lowered, an appreciable quantity of albumen is present, with hyaline, granular, and epithelial casts, sometimes with red and white blood cells. If the number of blood cells is sufficient to color the urine, the patient's attention is attracted by the change in color; the diminished quantity he is apt not to notice. These changes in the urine last for four or five weeks and then disappear.

CASE VIII. *Mild Exudative Nephiritis; Three Attacks, with Complete Recovery.*—A physician, in May, 1868, while serving as interne in a hospital, felt rather miserable for about three weeks, and had a good deal of albumen, blood, blood casts, and hyaline and granular casts in the urine. After the subsidence of the constitutional symptoms, the albumen continued in the urine for several weeks.

In October, 1868, he had a second attack of the same character, the albumen persisting for about two months.

After that his health was perfectly good and he had no albumen until January, 1875. Then he had an ordinary sore throat with fever. Albumen, blood, hyaline and granular casts were present in the urine. The constitutional symptoms subsided after a week, the albumen continued in small quantities for several weeks.

From that time up to the present—December, 1903—his health has been good and he has had no renal symptoms.

2. In a considerable number of cases of influenza, more frequently in children than in adults, there is developed an acute exudative nephritis of varying severity. The urine contains albumen in large quantity, casts and sometimes blood; its specific gravity is not lowered. The quantity in severe cases is diminished. In the mild cases there are no renal symptoms except the changes in the urine. In the severe cases there may be dropsy or convulsions.

As a rule, the nephritis is transitory and the patients recover inside of four weeks.

CASE IX.—Male, 21 years old, January 13, 1898. He was perfectly well until twenty-four hours before admission to the hospital. Then he began to have general pains, frontal headache, coryza and fever.

January 13: Temperature 102°, pulse 96, breathing 28. The urine is of a specific gravity of 1.030, contains a large quantity of albumen and many granular casts.

January 16: There is no more albumen and no casts in the urine. The urea is nine grains to the ounce.

CASE X.—Female, 19 years old, January 24, 1898. For the past month she has felt tired, weak and headachy, has looked pale and has lost flesh and strength.

Three days ago she had to stop work on account of fever, had headache and general pains.

January 24: Temperature 101°, pulse 140, breathing 28. The specific gravity of the urine is 1.034, the quantity 20 ounces; it contains a great deal of albumen and many hyaline and granular casts. The hæmoglobin is 52 per cent. The legs are œdematous. It is evidently a case of simple anæmia complicated by influenza.

February 2: The albumen and casts have disappeared from the urine, the dropsy has gone, the influenza has run its course, the anæmia still persists.

3. The ordinary cases of acute exudative nephritis, occurring with scarlatina, diphtheria or without discoverable cause, vary in their severity, but all give characteristic symptoms. The only diseases with which they can be confounded are: acute productive nephritis and exacerbations of a chronic nephritis.

The quantity of urine is diminished at the outset of the nephritis, and continues small until the activity of the inflammation has subsided; then the quantity increases from day to day and may even exceed the normal. The quantity of the urine must be in proportion to the quantity of the blood which passes through the kidneys, so that this quantity gives us a measure of the intensity of the congestion which is arresting the circulation of the blood through the kidney. Complete suppression of urine is a serious symptom, both because it denotes an intense nephritis and because it is of itself a cause of death. The production of only a few ounces of urine in each twenty-four hours is the rule in a great number of cases, and is not necessarily of serious import; if it only last a few days the patients do perfectly well. If the scanty excretion of urine is kept up for a number of days, opinions vary as to the results. Some believe that the diminished quantity of urine is the cause of the dropsy. Some think that the deficient excretion of excrementitious substances causes the convulsions. Some believe that the principal effect of a diminished excretion of urine is to cause bodily feebleness.

Unquestionably, the production of urine may be very small for a number of days, and yet the patients do well. Dr. Whitelaw (*Lancet*, September 29, 1877) reports a case of anuria, lasting for twenty-five days. The patient was a boy eight years of age. The suppression of urine began twelve weeks after the beginning of a scarlet fever. With the exception of two ounces passed on the thirteenth day, there was complete anuria for twenty-five days. Except for slight headaches and later slight œdema, there were no uræmic or dropsical symptoms throughout. There was no albuminuria and no fever. The boy was watched night and day. He recovered entirely.

The specific gravity of the urine remains normal or is higher, while the quantity is small; when the quantity is increased the specific gravity falls a little.

The appearance of the urine is turbid, or smoke-colored, or bloody.

Albumen is present in very large quantities. Casts are numerous—hyaline, granular, nucleated, epithelial, and blood. There are also red and white blood cells, and epithelial cells from the kidneys and from the bladder. As a rule the quantity of albumen and the number of casts are in proportion to the severity of the nephritis, but this is not always the case. Large quantities of albumen, numerous casts, and many red and white blood cells may be found in the urine of kidneys which, after death, show no structural changes except in the glomeruli. While, on the other hand, small quantities of albumen and a few hyaline casts are compatible with a severe nephritis. Still further, the number of casts found in the urine during life is not always in pro-

Case XI. *Acute Exudative Nephritis with Scarlatina.*—A girl, 9 years old, had a mild attack of scarlatina. Desquamation was nearly completed when the child suddenly became short of breath, and by the next day had developed general subcutaneous dropsy. The urine was very scanty, it was nearly solid with albumen and contained hyaline, granular and epithelial casts. The child died with dyspnœa and dropsy, but without coma or convulsions, on the fifth day of the nephritis.

Autopsy. In the kidneys the epithelium of the convoluted tubes is swollen; the tubes contain coagulated and granular matter. In the Malpighian bodies the capsule cells and tuft cells are swollen, the capillaries are matted together with exudate.

Case XII. *Acute Exudative Nephritis: Puerperal Convulsions.* Female, 22 years old, primapara, 8 months pregnant, had been feeling perfectly well; went out to a supper party, was suddenly attacked with convulsions, and died at the end of 36 hours.

The urine was loaded with albumen and contained hyaline, granular and epithelial casts. The child was delivered dead.

Autopsy. The kidneys are somewhat enlarged, the cortex thick and pale.

In the Malpighian bodies the capsule and tuft cells are swollen.

In the convoluted tubes the epithelium is flattened; in the cavities of the tubes there is a good deal of granular matter. In the straight tubes of the cortex the epithelium is swollen, granular and partly detached. Some of the tubes contain casts.

In the pyramid tubes the epithelium is swollen, granular and partly detached. Some of the tubes contain cast matter.

Case XIII. *Acute Exudative Nephritis; Puerperal Convulsions.* Female, 28 years old.

October 2, 1890, at 12 noon, labor began. At 2:30 P.M. a convulsion; the child was delivered at once; 10 minutes later another convulsion. After this eighteen more convulsions, and she died the next day.

Ten days before labor the urine was normal; after delivery it contained a large quantity of albumen.

The temperature after delivery was 102° to 108°, the pulse 160 to 180.

Autopsy. The brain and its pia mater are congested and œdematous.

The lungs are covered with old pleuritic adhesions; at both apices there are old tubercles and fibrous tissue.

The right ventricle of the heart is hypertrophied, the valves are normal.

The liver is fatty and congested.

The spleen is normal.

The uterus is normal.

The kidneys are enlarged, the cortex pale; the epithelium of the convoluted tubes is much swollen; the glomeruli show a marked growth of the tuft cells.

CASE XIV. *Acute Exudative Nephritis; Endocarditis.*—Male, 39 years old, October 8, 1885. Six weeks ago he began to have dropsy of the legs, cough with muco-purulent sputum, dyspnœa, no headache and no vomiting.

Now he is fairly nourished, anæmic, pulse 104, respiration 32, temperature 97°, urine 1.011, a good deal of albumen, hyaline, granular and epithelial casts.

October 20: The heart's action is irregular; there is a double aortic murmur and a mitral presystolic murmur.

There are sibilant and sonorous râles over both lungs.

October 29: The dropsy and dyspnœa are increasing; he is delirious at night.

November 1, he died.

Autopsy.—Both lungs are congested, there are old pleuritic adhesions.

The aortic valves are thickened, ulcerated, and covered with large, soft vegetations. The mitral valve is thickened and stenosed.

The liver and spleen are large and firm.

The kidneys are large, their surfaces smooth; the cortex thick, white, mottled with red.

The epithelium of the straight tubes in the cortex is swollen; some of the tubes contain casts.

The epithelium of the convoluted tubes is flattened.

In the Malpighian bodies there is a marked growth of the tuft cells.

Treatment.—We have to treat an acute exudative inflammation of the kidneys, which naturally runs its course in four weeks and terminates in recovery. We have also to treat the symptoms of this nephritis—the scanty urine, dropsy, vomiting, anæmia, and cerebral symptoms. We have to treat these conditions more frequently in children than in adults, and very often as complicating an infectious disease.

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The aortic valves are thickened, ulcerated, and covered with large, soft vegetations. The mitral valve is thickened and stenosed.

The liver and spleen are large and firm.

The kidneys are large, their surfaces smooth; the cortex thick, white, mottled with red.

The epithelium of the straight tubes in the cortex is swollen; some of the tubes contain casts.

The epithelium of the convoluted tubes is flattened.

In the Malpighian bodies there is a marked growth of the tuft cells.

Treatment.—We have to treat an acute exudative inflammation of the kidneys, which naturally runs its course in four weeks and terminates in recovery. We have also to treat the symptoms of this nephritis—the scanty urine, dropsy, vomiting, anæmia, and cerebral symptoms. We have to treat these conditions more frequently in children than in adults, and very often as complicating an infectious disease.

The most efficient treatment of the nephritis is the application of heat to the entire surface of the body. This can be done in a number of ways, but the best way is to wrap the entire body in a blanket wrung out of hot water. Such a hot pack can be used for an hour at a time once or twice a day. Of drugs the most reliable is aconite—one or two minims of the tincture every hour. It may be necessary to precede the aconite by giving one drachm of sulphate of magnesia every hour until the bowels move, or until eight doses have been taken. There are cases, in which the nephritis is not of very acute type, where digitalis seems to exert a favorable effect on the circulation.

The scanty urine often causes anxiety. Of course it is better that the patients should pass a fair quantity of urine, but I think that there is a tendency to exaggerate the dangers of scanty urine and to be too energetic in giving diuretics. As the diminution in the quantity of urine is due to the congestion of the kidneys, if we can decrease the congestion the urine will increase. The best way to do this is to apply heat to the surface of the body. The use of diuretics is to be avoided. The attempts to make up for the scanty production of urine by purging or sweating the patient have never seemed to me to be of any practical use.

The febrile movement in an acute nephritis requires no treatment.

The prostration, loss of appetite, nausea, and vomiting only call for rest in bed and a fluid diet.

The anæmia ought to be prevented or relieved, but, while the nephritis is still active, I know of no way in which this can be done. When convalescence is established, then the anæmia readily improves with the ordinary methods of treatment.

The dropsy is subcutaneous, and even when considerable, does little harm. It disappears of itself as the nephritis subsides. The rest in bed and the hot pack are all the treatment necessary for it. To give diuretics or cathartics to get rid of the dropsy is quite useless.

The cerebral symptoms are the ones to which most attention has been directed. There can be no question that they accompany a contraction of the arteries with increased arterial tension, and labored action of the heart. No matter what views one may entertain as to the cause of this change in the circulation, I believe that treatment is best directed to the arteries themselves, rather than to the uncertain causes of their contraction. Fortunately there are drugs which stop contraction of the arteries promptly and efficiently. Of these drugs the most suitable are: aconite, nitro-glycerin, chloral hydrate, and opium, preferably given in small doses and at regular intervals before the cerebral symptoms are marked, but in large doses hypodermically or by the rectum to stop a severe attack.

It is wise to watch the condition of the heart and arteries, and as soon as increased arterial tension is developed, not to wait for the manifestation of the cerebral symptoms, but to try to relieve it at once.

The way in which we manage the patients, therefore, is as follows: They are put to bed or kept in the house until the nephritis has run its course. They are put on a fluid diet, preferably milk, and the skin of the entire body is cleaned once a day. For many cases no other treatment is necessary.

If vomiting is troublesome it can usually be controlled by adding oxalate of cerium and bicarbonate of soda to the milk. For the restlessness and sleeplessness chloral hydrate, the bromides, or opium may be employed.

If the nephritis is of severe type the patient is wrapped in a blanket wrung out of hot water and kept in it for one hour either once or twice every day. In addition we give a drachm of sulphate of magnesia every hour until the patient has taken eight doses or the bowels begin to move. This is followed by one or two minims of tincture of aconite every hour.

Throughout the disease we watch the pulse, and as soon as it shows any increased tension give chloral hydrate in doses of from two to five grains every three hours.

If severe headache, muscular twitchings, or general convulsions occur, to most of the patients we give chloral hydrate in doses of from 5 to 20 grains by the rectum, or nitro-glycerin in doses of from $\frac{1}{200}$ to $\frac{1}{50}$ of a grain hypodermically, or morphine in doses of from $\frac{1}{10}$ to $\frac{1}{6}$ of a grain hypodermically. In strong and robust adults with a good deal of venous congestion, general blood-letting may be advisable. For the relief of the convulsions urethane in solution, given in repeated doses up to 100 grains in twenty-four hours, is said to be of service.

As the nephritis subsides the milk is gradually replaced by solid food, and iron and oxygen are given.

The management of the mild cases of acute exudative nephritis is quite different. They require only ordinary care and need no drugs. They do best with ordinary food and out-door exercise.

ACUTE EXUDATIVE NEPHRITIS WITH AN EXCESSIVE PRODUCTION OF LEUCOCYTES.

Anatomically these cases can be classed with acute exudative nephritis, but clinically they belong to an entirely different group. They are to be classed with the primary streptococcus inflammations of the viscera. They are not to be confounded with embolic nephritis, nor with nephritis caused by cystitis.

The nephritis is seen both in children and adults. I have seen it with scarlatina, diphtheria, measles, and occurring without discoverable cause.

The invasion is sudden, with a high temperature and marked prostration. Restlessness, headache, delirium, and stupor are soon developed and continue throughout the disease. The papients rapidly lose flesh and strength and pass into the typhoid state. Dropsy is slight, or absent altogether. The urine is not so much diminihed in quantity as one would expect. Its specific gravity is not changed. Albumen, casts, and red and white blood cells are present in considerable quantities, but not always early in the disease, and they may even be absent altogether.

Although this form of nephritis is not of common occurrence, the unusual characters of the symptoms and the great mortality are reasons for calling special attention to it. If differs from the ordinary form of exudative nephritis in that it behaves like an infectious inflammation, and that, although the emigration of white blood cells is large, the exudation of serum may be small, and so the urine may show but little changes. It is probable that the nephritis is the result of some obscure form of bacteritic infection.

CASE XV. *Acute Nephritis, Septic Form.* Male, 64 years old, stableman, October 15, 1883. Five days ago he noticed that his feet were a little swollen and his urine dark colored.

Four days ago he became jaundiced, with pain over the epigastrium and dry retching.

Now, on the sixth day, he is emaciated, jaundiced, the stools white, urine 1011, contains bile but no albumen, pulse 96, respiration 20, temperature 97°.

Tenth day: The jaundice is fading, urine 1.013, no albumen, less bile.

Fourteenth day: A severe chill, but no fever.

Seventeenth day: He is dull and stupid, alternating with delirium, the urine is diminished in quantity, the tongue is dry and brown, the heart is rapid and feeble.

Eighteenth day, he died.

Autopsy.—The brain was not examined. The lungs are emphysematous, with pus in the bronchi.

The heart is small, the aortic valves a little thickened, the arch of the aorta is dilated and atheromatous.

The liver, biliary passages and gall bladder are normal.

The stomach shows the changes of chronic gastritis.

The intestines are normal.

The spleen is large.

The kidneys weigh together 24 ounces, the capsules are not adherent, the surfaces are smooth, the cortical portions are thick and intensely congested.

The Malpighian bodies are unchanged. The epithelium of the cortex tubes is flattened, there are large numbers of leucocytes infiltrated between the tubes, the capillary veins are large and full of leucocytes.

The epithelium of the pyramid tubes is degenerated, there is a moderate infiltration of leucocytes between the tubes, the large veins contain leucocytes.

CASE XVI. *Acute Nephritis, Septic Form.*—Female, 5 months old, was in the Foundling Asylum.

January 23, 1880, after having been in the asylum for two weeks, she was put out to board.

February 26 she was brought to the Out-Door Department, having been taken sick two days before. The temperature was then 104°, there were coarse and fine râles over both lungs.

After this the temperature ran from 102° to 104°; the child was constantly restless and moaning as if in pain. There was no vomiting and no convulsions. The urine was passed freely, but was not examined.

March 12 she died, on the sixteenth day of her illness.

Autopsy.—There are no lesions in any of the viscera except in the kidneys. The kidneys are large. There are no changes in the pelves of the kidneys, the ureter, or the bladder.

There are narrow segments beginning at the surface of the kidney and running through the cortex into the pyramids, which are of much lighter color than the rest of the kidney.

The changes in the kidney are diffuse, but are much more marked in the white segments.

The larger veins are distended with blood.

The epithelium of the tubes is in some places swollen, in some detached and necrotic, in some flattened.

The tubes contain necrotic epithelium, cast matter, coagulated matter, and many are filled and distended with leucocytes.

Some of the Malpighian bodies contain leucocytes.

There is a moderate infiltration of leucocytes between the tubules.

ACUTE PRODUCTIVE (OR DIFFUSE) NEPHRITIS.

Definition.—An acute inflammation of the kidneys, characterized by exudation from the blood vessels, a growth of new connective tissue in the stroma, and changes in the epithelium and the glomeruli.

Synonyms.—Acute Bright's Disease; Parenchymatous Nephritis; Croupous Nephritis; Glomerulo-Nephritis.

Etiology.—This is the most serious and important of the forms of acute nephritis, for the reason that its lesions are from the first of a permanent character. It does not follow exudative nephritis, nor is it merely a modification of it; from the very outset it is a different form of inflammation. In the kidneys of persons who have been sick only a few days, the characteristic lesions are already evident. Productive nephritis is governed by the same law as that which belongs to productive inflammation in other parts of the body—the disposition of the inflammation to continue as a sub-acute and chronic condition. It is of importance to recognize that in exudative nephritis the lesions are temporary, and after their subsidence the kidneys return to their normal condition, just as the lungs do after a lobar pneumonia. In productive nephritis, on the other hand, some of the lesions are permanent; the kidneys can never return to their normal condition, just as in an interstitial pneumonia the lung never gets rid of the new connective tissue.

Post-scarlatinal nephritis is nearly always of the productive form. Nephritis complicating diphtheria, or developed during pregnancy is very frequently of this type. A primary nephritis in a person over twelve years old, if of sub-acute form, is almost invariably a productive nephritis. On the other hand, this form of nephritis very seldom complicates any of the infectious diseases except scarlatina and diphtheria.

These facts assist very much in making the diagnosis between the two forms of acute nephritis. It is easy to remember that post-scarlatinal nephritis and primary nephritis of sub-acute type are nearly always of the productive form; and that nephritis with diphtheria and pregnancy is often of the productive form; while acute nephritis under all other conditions is regularly of the exudative form.

Morbid Anatomy.—The kidneys are increased in size, the capsules are not adherent, the surfaces are smooth. The cortical portion is red, or white, or mottled. The mucous membrane of the pelvis is sometimes congested. Of the tubules in the cortex, in some the epithelium is flattened, in some there is coagulated matter or casts, in some the epithelium is swollen, degenerated, or contains globules of fat. In those parts of the cortex where there is a growth of new connective tissue the tubes may be atrophied. The tubules of the pyramids show but little change except that they may contain casts. In the stroma of the cortex there is a growth of new connective tissue, varying in different kidneys as to the relative proportion of cells and basement substance. This new tissue in many of the kidneys follows the line of the arteries which run up into the cortex, so that it takes the form of wedges.

But in other kidneys the new tissue is diffuse, or in irregular patches.

Many of the glomeruli show only an increase in the size and number of the cells which cover the capillaries, with some swelling of the capsule cells. But in others there is an excessive new growth of capsule cells which compresses the tuft of vessels. This growth of new cells from the capsule cells must not be confounded with accumulations of white blood cells within the capsules, nor with the growth of new cells on the walls of the capillaries. The glomeruli which are changed in this way are in groups, each group corresponding to some one artery.

The whole picture of the nephritis is that of a combination of exudative and productive inflammation.

When such a nephritis becomes chronic it is often possible to follow its course for many years, and to see at the end of that time that the anatomical changes in the kidney are of the same kind, but much more extensive.

Symptoms.—Of the patients who suffer from this form of nephritis, a certain number behave as if they had a simple exudative nephritis. There is a rise of temperature with more or less prostration. Cerebral symptoms are marked—headache, stupor, sleeplessness, restlessness, muscular twitchings, and general convulsions. The arteries are contracted, the pulse is of high tension, the heart's action is exaggerated, the left ventricle may be hypertrophied, there is dyspnœa. The appetite is lost, there may be nausea and vomiting. The urine is scanty or suppressed, it is colored by blood, and contains much albumen and many casts. The patients are very sick and much more likely to die than they are with an exudative nephritis.

Such a nephritis may, however, apparently run its course. At the end of four weeks the symptoms subside and the patients get better. They may then remain in ordinary health without renal symptoms for weeks, months, or years. But sooner or later they have another acute attack, or they gradually develop the symptoms of a chronic nephritis.

CASE XVII. *Acute Productive Nephritis, Post-Scarlatinal.*—

Male, 22 years old, May 22, 1876. Five weeks ago he was attacked with scarlatina.

During convalescence he was attacked with general dropsy, loss of vision, nausea, and the urine was scanty and high colored.

Now there is general dropsy; pulse 96, temperature 100°, urine 10 ounces; a large quantity of albumen.

On June 1 and 2 he had several convulsions. The urine continued to be scanty, with much albumen and many casts of all kinds.

June 9, he died.

Autopsy.—The brain was not examined. Both ventricles of the heart are moderately dilated, the pericardial sac is distended with serum.

The lungs are œdematous and both pleural cavities contain large quantities of serum.

The stomach is congested and coated with mucus.

The liver, spleen and intestines are normal.

The kidneys are large, smooth, with a thick, white cortex.

In most of the cortex tubes the epithelium is flattened; some of the tubes contain cast matter.

In the Malpighian bodies there is a marked growth of the capsule cells, the tuft cells are swollen.

In the stroma of the cortex there is a diffuse growth of connective tissue, with scattered collections of small round cells.

In the pyramids the epithelium is swollen and detached; many of the tubes contain casts.

CASE XVIII. *Acute Productive Nephritis; Scarlatina.*—Female, 3 years old.

October 29, 1882, she became dull and languid and vomited.

October 31 she had the tongue and eruption of scarlatina.

November 3 the eruption was dusky colored.

November 5 temperature 104°; there was a discharge from the nose; the urine contained no albumen.

November 6 she began to desquamate; both ears were discharging, the throat was much swollen, temperature 103°.

November 6 there was discharge from the nose, false membrane on the pharynx, and the symptoms of laryngitis.

November 8 the urine contained no albumen or casts.

November 13 there were subcrepitant râles over both lungs.

November 16: Temperature 103°, no albumen or casts in the urine.

November 18: Urine diminished in quantity; 1.020; a large quantity of albumen, blood and granular casts; temperature 100°.

November 19: Vomiting; temperature 104°; urine scanty.

November 21: Urine almost suppressed; temperature 103°; delirium, restlessness, dyspnœa.

November 23, she died.

Autopsy.—There is moderate dropsy of both legs.

The brain and its membranes are normal.

The pharynx and trachea are coated with false membrane.

The lungs show the lesions of broncho-pneumonia.

The liver and spleen are normal. The kidneys are large, smooth, with pale cortex and red pyramids.

The cortex tubes are dilated, the epithelium is flattened; the tubes contain coagulated matter and cast matter. The Malpighian bodies are large and opaque, with a considerable growth of tuft cells.

Here and there in the cortex are long, narrow strips running parallel with the large vessels. In these strips the tubes have nearly disappeared and there is an extensive growth of connective tissue. In the Malpighian bodies the walls of the capillaries are thickened and the capsule cells are increased in number.

CASE XIX. *Acute Productive Nephritis; Primary.*—Male, 40 years old, January, 1879. One month ago he was taken sick with headache, bronchitis, dropsy of the legs, scanty and bloody urine.

For four days he has been much worse, with dyspnœa, general dropsy; the urine scanty, containing blood, much albumen, and many hyaline, granular, and epithelial casts.

Autopsy.—At the apices of the lungs there are old miliary tubercles; the lungs are œdematous.

The liver is cirrhotic.

The kidneys are large, smooth, with a thick pink cortex.

In the Malpighian bodies the capsule cells are swollen, the tuft cells are multiplied.

In the cortex tubes the epithelium is flattened and granular; the tubes contain cast matter.

In the pyramid tubes the epithelium is swollen, or flattened, or detached. The tubes contain casts, blood and leucocytes.

In the stroma there are many patches of connective tissue.

CASE XX. *Acute Productive Nephritis; Puerperal.*—Female, 45 years old, December 5, 1881. She has had four children and has considered herself strong and well until two months ago. Then, while nursing her last child she was suddenly attacked with dyspnœa, headache, cardiac palpitation, general dropsy; the urine scanty and smoky.

She continued to get worse, with headache, dyspnœa and dropsy; the urine varied in quantity from 1 drachm to 6 ounces, and contained much albumen but few casts, sometimes blood.

She became anæmic, developed muscular twitchings and coma, and died on January 11.

Autopsy.—There is an increase of clear serum beneath the pia mater of the brain, and the pia mater itself is œdematous. The brain tissue is not œdematous.

The lungs are congested and œdematous. The walls of the heart are normal, the mitral valve is thickened and stenosed. The aorta and large arteries are normal. The liver and spleen are firm. The stomach and small intestines are congested.

The kidneys are of medium size, the capsules slightly adherent, the surfaces finely nodular.

In the cortex there is a considerable growth of connective tissue following the wedge type.

The epithelium of the cortex tubes is flattened and the tubes contain cast matter.

The capsule cells of the Malpighian bodies are increased in number.

CASE XXI. *Acute Productive Nephritis; Primary; Apparent Recovery.*—Male, 36 years old, November 15, 1882. Four weeks ago he was exposed to cold and wet. Then he had irregular chills, a little œdema of the legs, the urine was scanty and dark colored.

Now there is general dropsy, dyspnœa, cough and mucous sputum, mild delirium, no vomiting or headache; temperature 98.6°, pulse 120, urine 1.019, much albumen, a little blood, hyaline, granular and epithelial casts.

November 17: Urine 10 ounces; delirium continues.

November 20: Urine 60 ounces.

November 27: Urine 66 ounces; no dropsy; out of bed.

December 2-21: Urine 85-110 ounces; no albumen, no casts.

The man seems to be well.

The more ordinary cases have a gradual invasion, and run a subacute rather than an acute course.

In some of the patients at first there are only loss of appetite, headaches, and an increasing pallor of the skin and mucous membranes; the dropsy does not come on until after many days.

In some of the patients dropsy of the legs is the first, and for a time the only, symptom. They continue to eat well, feel well, and attend to their work.

In most of the patients dropsy of the legs and face, anæmia, headache, sleeplessness loss of appetite, nausea and vomiting are developed at about the same time.

The urine is only moderately diminished in quantity; it often contains no blood, there is a large quantity of albumen and a considerable number of casts. The specific gravity remains normal, or falls a little.

The course of the disease varies in the sub-acute cases.

1. The patients have an attack lasting for several weeks, characterized by albumen and casts in the urine, dropsy, anæmia, headache, and disturbances of digestion. These symptoms may be well marked or slight. It is not uncommon to see a child going to school while suffering from nephritis.

The attack runs its course, the patients get well, but they get well with damaged kidneys. They do not continue to have a nephritis, but they have kidneys which are susceptible to fresh attacks of inflammation. Sooner or later, it may not be until after several years, the patients may have a chronic nephritis.

2. The patients begin in the same way with an attack which lasts for several weeks. Then the symptoms subside, but the nephritis seems to continue as a very slow chronic process with few symptoms. The urine usually contains a little albumen and a few casts. The excretion of urea may be somewhat diminished. There is a disposition to secondary anæmia. But the general health may remain good even if they work hard and lead an ordinary life.

But in other patients the excretion of urea is considerably diminished, the general health is never very good; they may have an exacerbation of the nephritis, or pass into the condition of chronic uræmia.

3. The first attack is followed by other attacks which follow each other at intervals of weeks or months. Between the attacks the patients are comparatively well. But each attack is worse than the preceding one, and finally there comes an attack which proves fatal.

4. The most distressing cases to witness are those which are severe and continuous. The patients are constantly getting worse, and yet months may elapse before their sufferings are terminated by death. The color of the skin and of the mucous membranes becomes more and more white; headaches are constant and troublesome; sleep is difficult and unrefreshing; the eyesight is impaired or lost altogether; there is no appetite, but rather constant nausea and irritability of the stomach; from time to time the arteries are contracted and there is a disposition to muscular twitchings and general convulsions. The dropsy constantly increases, no matter how large the excretion of urine. The subcutaneous connective tissue is everywhere œdematous and the serous cavities are filled with serum. It seems as if the blood serum was unable to remain in the vessels; it escapes everywhere.

Not only does treatment fail to control the nephritis, but we are unable even to alleviate the symptoms.

Prognosis.—The majority of cases of acute productive nephritis terminate unfavorably. Either the disease goes on continuously and the patients die at the end of a few days or a few months; or the acute symptoms subside and a chronic nephritis supervenes. It is not wise, however, to give too unfavorable a prognosis even in severe cases; great improvement and even complete recovery are possible. I see from time to time persons, in apparently good health and able to earn their living, concerning whom I have given a very unfavorable prognosis many years ago.

CASE XXII. *Sub-Acute Productive Nephritis; Primary; Early Stage; Death from Lobar Pneumonia.*—Male, 34 years old, May 2, 1884. One month ago he began to have dropsy, first of the legs, then of the scrotum; vomiting, dyspnœa, with dark-colored urine.

Now, pulse 100, breathing 30, temperature 104°. Urine 4 ounces, 1.012, large quantities of albumen, blood and epithelial casts. The legs are dropsical.

There are dullness and bronchial voice and breathing over the upper lobe of the right lung.

He went on with the symptoms of the lobar pneumonia of the right lung until May 6. Then it was evident that pneumonia was beginning in the left lung; he became delirious and died on May 7.

Autopsy.—There is general dropsy. The heart is normal.

The upper and middle lobes of the right lung are in the condition of red hepatization. Both lobes of the left lung are in the early stages of red hepatization.

The liver, spleen and intestines are normal. The stomach shows the lesions of chronic gastritis.

The kidneys weigh together 16 ounces; their surfaces are smooth, the cortical portion thick, mottled red and gray.

The epithelium of the cortex tubes is flattened; a part of the tubes contain cast matter.

Through the cortex there are narrow strips, parallel to the large vessels, where there is growth of connective tissue, atrophy of the tubes and a growth of the capsule cells of the Malpighian bodies.

CASE XXIII. *Sub-Acute Productive Nephritis; Primary; Three Months' Duration.*—Male, 38, plasterer, January 29, 1882. He was always strong and well until three months ago. Then he began to vomit and have headaches. After three weeks he began to have general dropsy.

For the past eight weeks he has had cough and muco-purulent sputum. The urine has been dark colored, but not diminished.

Now he is emaciated, anæmic, a little dropsy of the face and legs; pulse 108, breathing 28, temperature 102° ; coarse râles over both lungs; urine 1.018, albumen, blood, granular casts.

After this the quantity of urine varied from 10 to 94 ounces, always containing albumen and casts. He passed into the condition of chronic uræmia and died on February 25.

Autopsy.—The body is emaciated. There is an increase of clear serum in the pia mater of the brain.

The left ventricle of the heart is moderately hypertrophied, the valves are normal.

There are a small number of old and recent miliary tubercles in both lungs.

The stomach and small intestine are coated with mucus.

The liver and spleen are normal. The kidneys weigh together 2 pounds; their surfaces are smooth, the cortex thick, mottled gray, white and red.

In the straight tubes of the cortex the epithelium is swollen; some contain cast matter, some blood.

The epithelium of the convoluted tubes is in some places flattened, in others swollen and detached; some of the tubes are dilated.

In the Malpighian bodies the tuft cells are increased in size and number; the capsule cells are swollen. There are patches of connective tissue between the tubes.

CASE XXIV. *Sub-Acute Productive Nephritis; Primary; Symptoms Continuous for Four Months up to Death.*—Female, 18 years old, October 9, 1877. Three months ago her feet and eyelids became swollen, and this swelling has continued up to the present time. She also had dyspnœa and vomiting and the urine was scanty and dark colored. The dropsy has continued and she has become very pale.

Now, October 9, there is general dropsy; urine 1.018, a good deal of albumen, no casts.

October 11: For twenty hours she passed no urine, then, after a hot-air bath, she passed 20 ounces.

After this the dropsy continued; vomiting and diarrhœa could not be controlled; there were no cerebral symptoms; the urine was abundant in quantity.

November 3 she died. There was general subcutaneous dropsy.

Autopsy.—The brain was not examined. The wall of the left ventricle of the heart is a little thickened, the wall of the right ventricle is thinned, the valves are normal.

Both pleural cavities are half-full of serum; the lungs are small and dry.

The liver and spleen are large and firm.

The stomach and intestines are normal. The kidneys weigh together 20 ounces. Their surfaces are smooth, the cortex thick and white, the pyramids red.

The convoluted tubes are dilated, the epithelium is flattened; there are casts and broken-down epithelium in some of the tubes.

In the Malpighian bodies the capsules are thickened and the capsule cells are increased in number so as to compress the tufts.

There is a diffuse growth of connective tissue between the cortex tubes.

CASE XXV. *Sub-Acute Productive Nephritis; Primary; Interval of Improvement.*—Female, 15 years old, January 3, 1883. She was perfectly well until one year ago. Then, after exposure to cold and wet, she suddenly developed dropsy of the face and legs, with slight cough and dyspnœa and scanty urine.

The dropsy increased and she entered a hospital where she remained five months. There she improved and was pretty well until two months ago, when the dropsy returned with headache, nausea and loss of vision.

She is now very anæmic, with general dropsy, dyspnœa, sibilant and subcrepitant râles over both lungs.

January 10, 1884, she is out of bed, with much less dropsy.

February 1: The dropsy has returned, with headache, vomiting and dyspnœa. Urine 30 to 57 ounces, 1.020-1.024, much albumen and many casts.

February 25 she died.

Autopsy.—All the viscera except the brain were examined and found normal, except the kidneys.

The kidneys are large, their surfaces smooth; the cortex thick and mottled red and white.

The epithelium of the cortex tubes is swollen and degenerated. There is an extensive growth of connective tissue between the tubes. In the Malpighian bodies there is a marked growth of capsule cells.

CASE XXVI. *Sub-Acute Productive Nephritis; Puerperal; Followed by Chronic Nephritis and Death at the End of Five Years.*—Female, 30 years old. February 8, 1881, she had a child; the labor was normal.

March 7, 1881, she went to bed well. She woke in the morning with headache, vomiting and fever. This was followed by the development of an abscess in one breast. The urine was dark colored but not diminished in quantitiy.

She was in bed for four weeks, the vomiting continuing during that time. After getting up she noticed there was dropsy of the face, feet and legs.

April 15 to July 14 she was up and about, complaining only of loss of appetite and dropsy of the legs.

July 14 to September 3 she was in a hospital, but did not improve.

October 13 she was anæmic, a little dropsy of the legs, increased arterial tension, a good deal of albumen and many casts in the urine.

February 24, 1882, she seemed to be well, with no albumen or casts in the urine. After this she seemed to be well and went through two confinements without any trouble.

April 14, 1886, she began to have headache, loss of eyesight, occasional vomiting and dyspnœa.

April 21 she came back to the hospital feeble, emaciated and anæmic; a friction sound over the heart; urine 1010, albumen, hyaline casts; urea 11 grains to the ounce.

She passed into the condition of chronic uræmia, and died April 25, 1886.

Autopsy.—The body is emaciated without dropsy. The lungs are œdematous. The pericardium is coated with fibrin. The mitral valve is thickened; the left ventricle is hypertrophied.

The liver and spleen are congested. The stomach and small intestine are congested and coated with mucus. The kidneys weigh together 6 ounces; the capsules are adherent; the surfaces are nodular.

Some of the cortex tubes are atrophied, others dilated; there is an extensive growth of connective tissue between the tubes; many of the Malpighian bodies are atrophied.

Treatment.—In the cases in which the disease behaves like an acute exudative nephritis the indications for treatment are the same as in the latter disease, although the results are not as satisfactory.

The management of the sub-acute cases requires the exercise of intelligence and care. It is necessary to appreciate exactly the condition of each patient. We notice the quantity of albumen, the number of casts, the excretion of urea, the presence or absence of dropsy, the degree of the secondary anæmia, and the general condition of the patient.

If a patient has little or no dropsy, no headache, no dyspnœa, no hypertrophy of the left ventricle, no chronic arteritis, no vomiting, and the hæmoglobin up to 70 per cent., we can disregard a good deal of albumen and many casts in the urine. These patients need solid food, an out-of-door life in a suitable climate, and no drugs except for the relief of functional disturbances. I even have very good results with persons who are obliged to live in cities and continue at their regular work.

The more severe cases have to be managed differently.

At first it is wise to keep the patients in bed and on an exclusively milk diet. In some of the patients the daily use of the hot pack seems to be of service; in some nothing is gained by its use; in some it is, I think, harmful. I do not know how to distinguish the appropriate cases for the hot pack except by trying its use for a few days.

Digitalis in small doses seems in some of the cases to exert a favorable effect on the nephritis. At all events the quantity of the albumen in the urine diminishes and the patients improve; but in some cases it does nothing. In the same way morphine in small doses, sometimes not more than $\frac{1}{50}$ of a grain, relieves the headache, sleeplessness and nausea, and the patients are evidently better for it. But there are other patients to whom the morphine is of no service whatever.

The dropsy is always of consequence. It is associated with a soft pulse; a faint heart action, rather feeble than forcible; no great disposition to venous congestion. The composition of the blood is profoundly changed by the diminution in the quantity of hæmoglobin and the number of red blood cells, and probably in other ways which we do not appreciate. The quantity of urine may be either diminished or increased. The dropsy, therefore, does not seem to depend on changes in the blood pressure or on the quantity of urine, but rather on changes in the composition of the blood or in the walls of the arteries. It is a dropsy which it is very difficult to treat intelligently.

There are cases in which the dropsy will disappear simply with the rest in bed and the milk diet.

There are cases in which profuse sweating by the hot-air bath or the hot pack diminishes the dropsy. But some of these patients cannot be made to sweat, some of them are too much depressed by the heat, in some the sweating does not diminish the dropsy.

Hydragogue cathartics, such as jalap and elaterium, will often diminish the dropsy for a time. Their use, however, cannot be continued for any length of time without irritating the stomach and intestines.

The daily use of good massage with compression of the legs by bandages is sometimes of real value.

Digitalis, caffeine, and strophanthus in many cases act efficiently, even when they do not increase the quantity of urine. They are the most useful drugs for this particular purpose.

The use of diuretics is limited to the cases in which the quantity of urine is diminished. In a patient with increasing dropsy, who is already passing 90 or 100 ounces of urine a day, diuretics are not indicated. The drugs ordinarily employed to effect diuresis are those which act on the circulation—digitalis, caffeine, strophanthus, and convallaria; and those which are supposed to act on the kidneys—acetate of potash, lactate of strontium, squills, diuretin. Good results are reported from the use of those drugs. But experience shows that in some patients the quantity of urine cannot be increased, and in others the increase in the quantity of urine is not followed by diminution of the dropsy.

It would seem as if the disposition to dropsy could be controlled if we could control the composition of the blood, increase the number of the blood cells, and raise the specific gravity of the blood serum. Unfortunately we do not know how to do this.

When necessary, to make the patient more comfortable, we have to tap the peritoneal and pleural cavities and to puncture the skin of the legs.

If the patients improve, the milk diet is to be gradually replaced by solid food, iron is to be given in fair doses, and the patient gets out of bed and out of the house. At this time climate becomes a matter of much importance. The patient should be sent to a warm, dry, equable climate, where he can lead an out-door life.

Chronic Congestion of the Kidneys.

There are a number of morbid conditions which interfere with the circulation of the blood in the aortic system in such a way that the blood accumulates in the veins and is diminished in the arteries. The most common of these conditions are: chronic inflammation of the aortic and mitral valves, dilatation of the heart, aneurism of the arch of the aorta, pulmonary emphysema, and large accumulations of fluid in the pleural cavities.

In pulmonary emphysema the disturbances of circulation are confined to the cases in which there is obstruction to the passage of blood through the lungs, dilatation and hypertrophy of the right ventricle, and then venous congestion. More or less dropsy is regularly developed at about the same time as the congestion of the kidneys.

Large accumulations of fluid in the pleural cavities, if they remain for any length of time, may produce well-marked chronic congestion. This is denied by Bartels, who says that he has never known congestion or any serious disturbance of the renal functions to occur as a result of pleuritic exudations. I have, however, seen congestion produced in this way a number of times. I think that it is a lesion of consequence to the patient, and I believe that it furnishes an additional reason for the early removal of fluid from the pleural cavities.

Etiology.—By far the most common cause of chronic congestion of the kidneys is disease of the heart. So long as a heart with chronic endocarditis, or myocarditis, or dilatation, is able, in spite of its damaged state, to carry on the circulation fairly well, no secondary changes in the kidneys are produced. But as soon as the blood accumulates in the veins to any considerable extent the kidneys suffer. One of three things regularly happens to them: either chronic congestion, or chronic degeneration, or chronic nephritis is developed. It is also necessary to remember that chronic endocarditis and chronic nephritis often exist in the same person, although neither one of them is secondary to the other.

Morbid Anatomy.—The kidneys are of medium size, or rather large. Their weight is increased, somewhat out of proportion to the increase in size. The color is dark red, the consistence is very hard, the surfaces are smooth, the capsules are not adherent. The congestion is most marked in the veins of the pyramids; they contain an increased quantity of blood, and are often dilated. The capillaries of the vortex are also congested, but it is rather exceptional to find them dilated. The epithelium of the convoluted tubes is swollen, and the separate cells of which it is composed are more evident. Or, instead of this, the epithelium is much flattened so that the lumen of the tube is larger. I think that this flattening of the epithelium belongs to the kidneys which give urine containing a good deal of albumen.

The most constant and characteristic change is in the glomeruli. The capillaries which make up the glomerulus are dilated, with more or less thickening of their walls. So far as I know this change in the glomeruli is constant and persists, even if the congestion is succeeded by a true nephritis.

While the congestion often persists up to the time of the patient's death, it may, instead, be followed by a chronic nephritis.

This may be a chronic nephritis either with or without exudation.

Symptoms.—Of the persons who die with chronic congestion of the kidneys a large number present marked symptoms during life, but it is difficult to determine how largely these symptoms are due to the congestion of the kidneys.

A congestion of a few weeks' duration, such as we see with large effusions of fluid in the pleural cavities, seems to give no renal symptoms and no changes in the urine.

The question is complicated by the fact that, with chronic endocarditis and pulmonary emphysema, any one of the following kidney lesions may co-exist:

1. Chronic congestion.
2. Chronic congestion followed by chronic exudative nephritis.
3. Chronic congestion followed by chronic nephritis without exudation.
4. Chronic degeneration.
5. Chronic degeneration followed by chronic nephritis with exudation.
6. Chronic nephritis with exudation not preceded by congestion.
7. Chronic nephritis without exudation not preceded by congestion.

Here are seven different kidney lesions, each one of them frequently associated with cardiac disease.

In bad cases of heart disease the ordinary symptoms are: dyspnœa, cough and expectoration, general dropsy, vomiting, headache, delirium, convulsions, coma, anæmia, loss of flesh and strength.

It is always difficult, and often impossible, in any given case of heart disease to determine certainly which kidney lesion exists. The general rules which we follow are these:

Chronic Congestion.—The cardiac symptoms not as severe;

dropsy often present; the quantity of the urine moderately diminished, its specific gravity normal or higher than normal, albumen in very small quantities, few or no hyaline casts.

Chronic Congestion followed by Chronic Nephritis with Exudation.—The cardiac symptoms more severe; the dropsy well marked; the urine scanty, its specific gravity normal or lowered, a large quantity of albumen, and casts in variable number.

Chronic Congestion followed by Chronic Nephritis without Exudation.—The patients are liable to have attacks of contraction of the arteries. The dropsy is not as constant. The quantity of the urine varies, sometimes above, sometimes below the normal. Its specific gravity is low. There is but little albumen except when the arteries are contracted. Loss of flesh and strength are marked features.

Chronic Degeneration.—The cardiac symptoms are severe; dropsy is regularly present but not always marked. The quantity of the urine varies, its specific gravity is unchanged, there is but little albumen. The patients are feeble and anæmic, with a prolonged period of scanty urine, delirium, stupor, and the typhoid state.

Chronic Degeneration followed by Chronic Nephritis with Exudation.—The cardiac symptoms are severe and the dropsy well marked. The quantity of urine is small, its specific gravity is rather high than low. Albumen is present in large quantities, casts are not as constant. The patients are feeble, anæmic, and get worse rapidly.

Chronic Nephritis either with or without Exudation, not preceded by Congestion.—In these patients there is the ordinary history of chronic nephritis lasting for months or years. Then, as the endocarditis advances, the heart's action becomes worse and the circulation is disturbed, the different cardiac symptoms are added.

Treatment.—Although we are unable to remove the mechanical interference with the circulation, yet the condition of the patients can often be very much improved. The main indications are to improve the character of the circulation and to remove the dropsy. In order to improve the circulation it is absolutely necessary to study the character of the heart's action, to appreciate the organic changes in the heart, and to determine whether the walls of the arteries are thickened, and whether there is an increase or diminution of the arterial tension. It is unfortunate that there is no instrument to tell us certainly what the arterial tension is; we have to depend upon the sense of touch. This, however, can be educated, and a physician is not competent to manage cardiac cases if he cannot appreciate changes in arterial tension.

In many patients with an irregular heart, dyspnœa and dropsy, simple rest is of the greatest service. This is especially marked in hospital patients who have tried to work up to the time of their admission. It is often a good rule to give no medicine to such patients until we see how much they improve with rest alone.

The regulation of the food is of importance. It is to be remembered that, while gastric indigestion and flatulence make the heart action worse, on the other hand meat is one of the best cardiac stimulants. Most patients are better for meat in some form at least once a day.

Tumultuous and exaggerated heart action may be due to contraction of the arteries and require such drugs as nitro-glycerin or chloral hydrate. It may be the expression of a heart which is really feeble and requires digitalis. It may mean a real exaggeration of the muscular action of the heart and be benefited by aconite, belladonna, and the local action of cold. A feeble heart without contraction of the arteries calls for cardiac stimulants; digitalis, strophanthus and caffeine are the best. But it is to be remembered that with aortic and mitral stenosis, myocarditis and diseased coronary arteries, there comes a time when cardiac stimulants make the heart's action worse instead of better. This is the time for the use of opium. Small doses of codeine or of morphine give the patient more relief than can be obtained in any other way.

In many patients the condition of the heart and arteries varies from day to day, so that there have to be many corresponding changes and combinations of the drugs—the cardiac stimulants, the arterial dilators, and the opium.

The dropsy is more or less favorably affected by the rest in bed and the regulation of the circulation. It can also be reduced by the hot pack and the hot-air bath.

For very extensive cardiac dropsies, with only chronic congestion of the kidney or but little nephritis, calomel is the most efficient diuretic. Three grains of calomel with $\frac{1}{16}$ of a grain of morphine four times a day for four days is the dose. The mouth and gums are to be kept very clean. If the drug acts properly there is but little effect on the bowels and no salivation, but on the third day the quantity of urine increases and remains large for several days. The dropsy diminishes and may disappear altogether.

CHRONIC DEGENERATION OF THE KIDNEY.

Definition.—A chronic disease of the kidneys characterized by degenerative changes in the renal epithelium.

Synonyms.—Chronic Bright's Disease, Chronic Parenchymatous Nephritis, Fatty Kidney.

Etiology.—The same mechanical obstructions to the circulation —heart disease, pleuritic effusions, etc.—which produce chronic congestion, can, instead of this, produce chronic degeneration of the kidney.

It is said that anæmia of the kidneys produces degeneration of the renal epithelium. Experiments upon animals show that this view is theoretically possible. It may be that the degeneration of the kidneys seen in old and feeble persons is due to a diminished blood supply, but we can hardly speak with certainty on this point.

Chronic diseases, such as phthisis and cancer, are followed by chronic degeneration of the kidneys.

There is a group of cases in which, although the health of the patients is not good, it is not easy to fix on a definite cause for the chronic degeneration.

Apparently many of the authors who describe a "chronic parenchymatous nephritis" include under this head both chronic degeneration and chronic nephritis.

The matter is further complicated by the fact that the kidneys may be in the condition of chronic degeneration for some time, and then become further altered by a chronic nephritis with exudation, and by waxy degeneration of the glomeruli.

Morbid Anatomy.—If the degeneration follow heart disease the kidneys are large, weighing together from sixteen to twenty ounces. Their surfaces are smooth; the cortical portion is thickened, of pink or white color, the pyramids are red. The gross appearance is that of the so-called large white kidney. The epithelium of the cortex tubes is swollen and coarsely granular. The capillaries of the glomeruli are dilated, with more or less thickening of their walls. The veins in the pyramids are congested. There are no changes in the stroma, or in the arteries.

If the degeneration follows phthisis, cancer, or any wasting disease, the kidneys are usually large, with a white or yellowish cortex. There are no changes except in the cortex tubes. In these the epithelial cells are either coarsely granular, or infiltrated with fat.

If the degeneration occurs in old people, or without discoverable cause, the kidneys may be either large and white, or of the size and appearance of a normal kidney, or small and red. There are the same degenerative changes in the epithelium of the cortex tubes, with no lesions in the stroma or the glomeruli.

Symptoms.—With the degeneration caused by heart disease the quantity of the urine varies with the changes in the action of the heart and the contraction of the arteries, sometimes abundant, sometimes scanty, sometimes suppressed. The specific gravity is not lessened, nor is the proportion of urea to the ounce diminished. Albumen and casts in small quantities are more frequently present than with chronic congestion. While it is difficult to separate the kidney symptoms from the heart symptoms, yet one has the impression that this kidney lesion is more serious than chronic congestion, and has its effect in increasing the loss of nutrition and the anæmia.

If the degeneration is followed by chronic nephritis with exudation, albumen appears in the urine in large quantities; the anæmia and dropsy are very marked, and the patients get worse rapidly.

When the degeneration accompanies chronic diseases, such as phthisis or cancer, the urine remains normal, or from time to time contains a little albumen and a few casts. The patients are so ill with their primary disease that the secondary degeneration of the kidneys is hardly appreciable during life.

The group of cases in which the degeneration occurs without discoverable cause is interesting, for the kidney lesion may be the cause of death. It is a difficult group to study, because the cases are not very numerous, and the clinical histories and autopsies are apt to be dissociated. The autopsies are for the most part in hospital patients with short and imperfect histories; while the clinical histories are of private patients on which it is difficult to obtain autopsies. I think, however, that it can be said the urine remains normal, or from time to time contains a little albumen and few casts. Neither dropsy, nor a pulse of high tension, nor an hypertrophied left ventricle, nor acute uræmic symptoms, are present.

The patients generally, month after month, lose flesh and strength, and become more or less anæmic. They may have a variety of digestive disturbances. The course of the disease is slow, sometimes interrupted by periods of improvement, but regularly getting worse from year to year. Finally the patients are so feeble that they remain in bed; they develop alternating delirium and stupor, and so die.

The diagnosis is these cases is extremely difficult. They resemble cases of malignant disease in which no tumor can be found, and cases of chronic nephritis with normal urine and no changes in the heart or arteries.

Treatment.—It is not easy to find means to influence chronic degeneration of the renal epithelium. The best that we can do for the patients is to stop all vicious habits, to regulate the diet and mode of life, and to relieve the disturbances of circulation.

CASE XXVII. *Chronic Degeneration of the Kidney, with Pul-*

monary Phthisis.—Male, 33 years old, November 11, 1875. He has had a cough for about a year. Three weeks ago he had a chill, followed by fever, headache, general pains, vomiting and diarrhœa.

Now, pulse 126, temperature 102°, urine 1018, no albumen; he is delirious and in the typhoid state. Over both lungs in front and behind there are dullness on percussion and subcrepitant râles. He continued in the same condition, and died on November 20.

Autopsy.—The brain was not examined. In the upper lobe of the right lung there is fibrous tissue, large cheesy nodules, and complete red hepatization. In the lower lobe there are scattered patches of red and cheesy hepatization. In both lobes of the left lung there are scattered patches of red and cheesy hepatization. The liver is normal. The spleen is large and soft.

In the lower part of the ileum there are a few small, tubercular ulcers. The kidneys are large, with thick cortex. The epithelium of the cortex tubes is markedly swollen and degenerated. The tuft cells of the Malpighian bodies are swollen.

CASE XXVIII. *Chronic Degeneration of the Kidney, after Childbirth.*—Female, 20 years old, December 7, 1874. Five months ago she was confined; since then she has complained of dyspnœa and cardiac palpitation.

For two months she has had a cough, with yellow, tenacious sputum, tinged with blood, and night sweats. She has also had headache, pain after eating, nausea and vomiting, but no dropsy.

Now there are no physical signs over the chest, no dropsy, urine 1018, contains no albumen.

December 9 she became delirious and had a general convulsion.

December 13 she died comatose.

Autopsy.—The brain is soft and œdematous. The heart is normal but rather soft. The lungs are small, congested; there is no pus in the bronchi.

The liver is fatty, the spleen is normal. The stomach and intestines are normal. The kidneys are of medium size, with smooth surfaces.

There is very marked swelling and degeneration of the epithelium of the cortex tubes. The appearance is like that of arsenic poisoning. The tuft cells of the Malpighian bodies are swollen.

CHRONIC PRODUCTIVE (OR DIFFUSE) NEPHRITIS WITH EXUDATION.

Definition.—A chronic inflammation of the kidney attended with a growth of new connective tissue in the stroma, permanent changes in the glomeruli, degeneration of the renal epithelium, exudation from the blood vessels, and sometimes changes in the walls of the arteries.

Synonyms.—Chronic Bright's Disease; Chronic Parenchymatous Nephritis; Chronic Glomerulo-Nephritis; Waxy Kidney; Large White Kidney; Chronic Diffuse Nephritis; Chronic Desquamative Nephritis.

It has been customary to hold that in all these kidneys the primary and most important changes are in the renal epithelium, while in another set of kidneys the primary and important changes are in the stroma. In other words, that the cases of chronic nephritis can be divided into two classes—parenchymatous nephritis and interstitial nephritis.

I do not think that this classification is supported by facts.

In all the forms of chronic nephritis changes are to be found in the renal epithelium, the glomeruli, and the stroma. Whether the changes in the stroma, the glomeruli, or the epithelium are the more marked makes no difference in the clinical symptoms. But the presence or absence of exudation from the renal blood vessels does correspond to a marked difference in the symptoms. The existence of the exudation from the renal vessels is easily shown by the presence of serum albumen in the urine. In this way we readily distinguish two forms of chronic nephritis, one with exudation and one without.

The way of looking at the matter, then, is this:

We find after death from chronic nephritis a great many varieties in the gross appearance of the kidneys. Some are large, some are small, some are red, some are white, etc. There is no regular correspondence between these different gross appearances of the kidneys and the clinical symptoms.

We find in these same kidneys changes in the renal epithelium, in the stroma, in the glomeruli, and in the arteries. Sometimes one, sometimes the other of these elements of the kidneys is the most changed. There is no regular correspondence between the predominance of the changes in one of the kidney elements over the other and the clinical symptoms.

On the other hand, the whole behavior of patients suffering from the two forms of chronic nephritis is so different that we must divide them into at least two classes. Until we can discover the causes which produce these forms of nephritis it is convenient to describe them according to the presence or absence of a disposition to the transudation of serum from the blood vessels.

In speaking of the exudation of serum from the vessels and its presence in the urine, we speak of it as it occurs during the whole course of the disease, and not as it occurs for short periods. We mean that in an exudative chronic nephritis there is usually a large quantity of albumen in the urine, but that there may be periods during which the albumen diminshes or entirely disappears. In the same way, in a non-exudative nephritis there may be periods during which albumen is present in considerable quantities. Generally speaking, the character of the clinical symptoms will vary with the presence or absence of the albumen.

Etiology.—A considerable number of cases of chronic nephritis follow an attack of acute or sub-acute productive nephritis. The conditions of chronic congestion and chronic degeneration of the kidney are not infrequently followed by a true nephritis.

Syphilis, chronic tubercular inflammation of any part of the body, chronic endocarditis, and chronic suppurative inflammations are often complicated with chronic nephritis.

It is very difficult to find a satisfactory cause for the primary cases. There are many of these, especially in young and middle-aged adults. The nephritis is developed in a slow, insidious way in persons whose previous health had been good, and in whom no exciting cause is discoverable.

Morbid Anatomy; Gross Appearance of the Kidney.—There is considerable variety in the gross appearance of the kidneys. The types which I have seen most frequently are as follows:

1. Large white kidneys, weighing together 16 ounces or more, the capsule adherent or not, the surface smooth or nodular, the cortex thick and white, the pyramids large and red.

2. Large mottled kidneys. These resemble the large white kidneys in every respect except that the cortex, instead of being white, is mottled in a variety of ways with white, yellow, red, and gray.

3. Kidneys which resemble types one and two, but are not enlarged, the kidneys together not weighing over 9 ounces.

The majority of the kidneys in chronic nephritis with exudation follow these three types.

4. Small kidneys, weighing together not more than 5 ounces, the capsules adherent or not, the surfaces nodular, the cortex thin, atrophied, white, the pyramids rather large and red. These kidneys belong to persons who have the symptoms of chronic nephritis for many years, with periods of apparent recovery.

5. Kidneys which have the ordinary appearance and consistence of the chronic congestion due to heart disease, but in addition the capsules are adherent and the surfaces finely nodular.

6. Kidneys of different sizes—large, medium sized, and small, with adherent capsules and nodular surfaces. The cortex is gray, or gray mottled with red. The kidneys do not look at all like the large white kidneys. This is a type of frequent occurrence.

7. Kidneys which in their size, color, and general appearance are hardly to be distinguished from normal kidneys, except that their capsules are adherent.

8. Kidneys of small size, weighing not more than 4 ounces, with adherent capsules. The cortex is atrophied, red and irregular. These kidneys are found in persons who have given symptoms of kidney disease for a number of years.

It might naturally be supposed that such marked differences in the gross appearance of the kidneys would correspond to equally marked differences in the clinical histories and minute lesions. This, however, is not the case. The clinical histories are practically interchangeable, and the minute lesions are essentially the same.

Microscopical Appearances.—If we make vertical sections of the cortex of all these kidneys, no matter what their size or color, we get with a low magnifying power the same general picture. Instead of the uniform and orderly arrangement of tubes and glomeruli which we see in the normal kidney, the tubes seem to be obliterated in some places and dilated in others. There is a growth of fibro-cellular tissue in regular wedges, in irregular patches, or diffuse between the tubules.

If we examine the different constituents of the kidney in detail we find:

The tubes are in some places of normal size, in some places atrophied, in some places dilated. The atrophied tubes are in the patches of new connective tissue. The dilated tubes are not very large, nor do they form cysts.

The epithelium of the tubes is in some places merely flattened. These tubes are empty, or contain coagulated matter, casts, and red and white blood cells. In other tubes the epithelium is more or less swollen, sometimes so much so as to completely fill the tubes. In still other tubes the epithelial cells are swollen, their reticulum is very coarse, with large meshes, and they are infiltrated with fat. The kidneys vary as to which of these changes in the epithelium predominates, but all of them may be found in the same kidney.

The new connective tissue is in the form of wedge-shaped masses in the cortex which follow the line of the straight arteries and veins, or it is in irregular masses, or it is arranged diffusely so as to separate the tubes from each other. The longer the nephritis lasts, the greater is the quantity of new connective tissue. The relative proportion of basement substance and cells and the density of the basement substance vary in the different kidneys. The new tissue is well supplied with blood vessels.

The glomeruli are changed in several different ways:

1. There is a growth of the capsule cells in such numbers that they compress the tufts. The cells covering the capillaries are also increased in size and number. The capsule cells may finally be changed into connective tissue and the tufts become atrophied.

2. The glomeruli are of large size, the cells covering the capillaries are increased in number so that the outlines of the vessels are lost, but yet the capillaries are not compressed nor the glomeruli atrophied.

3. There is a growth of the cells which cover the capillaries

and of the cells within them. The cells which cover the capillaries become very large, the capillaries become obliterated and the glomeruli eventually become atrophied.

4. The walls of the capillaries become the seat of waxy degenerations, while the cells which cover them are increased in size and number.

5. If the nephritis follows chronic congestion of the kidney, the glomeruli remain large, the capillaries are dilated, with thickened walls, while there is added a growth of the cells which cover the capillaries.

The renal arteries behave in the same way as they do in non-exudative nephritis. In some cases they are unchanged; in others they are the seat of obliterating arteritis; in others there is a thickening of the muscular coat; in others there is thickening and sclerosis of the entire arterial wall; in still others there is waxy degeneration.

The glomeruli are the one part of the kidney which differs in the two forms of chronic nephritis. In nephritis with exudation the growth of the capsule cells, of the cells which cover the capillaries and of the endothelium which lines the capillaries is regularly well marked. In nephritis without exudation, as will be seen later, these changes are not as well marked, or are absent.

Symptoms.—The urine varies in quantity at different times in the course of the disease. In the earlier periods the urine is often scanty or even suppressed. If the disease goes on rapidly the quantity of urine may continue small; if it goes on slowly the quantity is often increased. In some of the worst cases, with general dropsy, the patients will pass more than one hundred ounces of urine a day.

With every exacerbation of the nephritis the quantity of urine is regularly diminished.

The specific gravity and the proportion of urea to the ounce of urine slowly diminish as the disease progresses. This is the rule, but there are exceptions to it. In cases which improve, the quantity of urea, after being much diminished, may increase until the patient excretes the full normal quantity for the twenty-four hours.

In the cases of shorter duration the specific gravity is apt to run between 1.012 and 1.020. In the very chronic cases it will be between 1.001 and 1.005. A very low specific gravity indicates a large growth of connective tissue in the stroma of the cortex, or waxy degeneration of the capillaries of the glomeruli and of the arteries of the kidney. Many persons who think that they have kidney disease get into the habit of drinking large quantities of mineral waters. This, of course, gives them urine of low specific gravity. In all doubtful cases it is necessary to determine the quantity of the whole excretion of urea for the twenty-four hours. There are patients in whom the quantity of urea is the principal factor in enabling one to decide between albuminuria without nephritis and chronic nephritis with exudation.

The urine regularly contains albumen and casts. During the active periods of the disease the quantity of albumen is very large; in the slow, prolonged cases the quantity is much smaller, and at times it may disappear altogether. Generally speaking, with large quantities of albumen the patients are dropsical and anæmic; with small quantities of albumen they are anæmic but not dropsical. There seems to be a common cause for the exudation of serum from the blood vessels throughout the entire body: the serum infiltrating the tissues, accumulating in the serous cavities, and mixing with the urine.

The number of casts is regularly in proportion to the quantity of albumen, but there are exceptions to this rule.

A peculiar pallor of the skin and white color of the sclerotic is seldom absent. This gives the patient a face very characteristic of chronic nephritis. In making a diagnosis in doubtful cases a good deal of importance is to be attached to the presence or absence of this appearance of the face. The change in the color corresponds to a diminution in the quantity of hæmoglobin and in the number of red blood cells. These changes in the blood are often not far advanced, but sometimes they are, and some patients even die with the symptoms of pernicious anæmia.

Dropsy may be considered almost a constant symptom of chronic exudative nephritis. There is an infiltration of the subcutaneous connective tissue with serum and an accumulation of serum in the serous cavities. The position of the fluid varies with that of the patient, accumulating in the dependent portion of the body. There is much variety as to the extent of the dropsy. In some patients there is never anything more than a moderate œdema of the legs, while in others a marked general dropsy is the most prominent symptom of the disease. There is also a variety as to the time of appearance and the duration of the dropsy. It may be one of the first symptoms of the nephritis, or it may not occur until late in the disease. When it is once established it may never leave the patient, or it may appear and disappear at irregular intervals.

Many of the patients are troubled with headache and sleeplessness. In some of them these symptoms exist only when the pulse is of high tension and disappear when the pulse becomes soft. In others, however, the headache and sleeplessness persist with a soft pulse. It must not be forgotten that these symptoms may also depend on digestive disturbances, and not on the disease of the kidneys.

Acute uræmic attacks, with contraction of the arteries, dyspnœa, vomiting, convulsions, etc., may occur at any time in the course of a chronic exudative nephritis. But they are of very much more frequent occurrence with the non-exudative form of the disease.

Chronic uræmia, on the contrary, is one of the ordinary ways in which an exudative nephritis proves fatal. The condition belongs to the later stage of the disease. It is developed rather gradually, but when once established is permanent, not disappearing up to the time of the patient's death. The patients are in a condition of alternating delirium and stupor, with a rapid, feeble, soft pulse.

Simple neuro-retinitis, or nephritic retinitis, may be developed at any time in the disease. Both eyes are regularly involved. The impairment of vision may be very slight, or considerable, or the patients may become entirely blind. With such a neuro-retinitis the prognosis of the nephritis is especially bad.

Dyspnœa is a nearly constant symptom, but it is not always the same kind of dyspnœa, nor produced by the same causes. It may be due to hydrothorax, to œdema of the lungs, to contraction of the arteries, or to failure of the heart's action.

The dyspnœa due to contraction of the arteries is common to both forms of chronic nephritis. It may be developed at any time during the course of the disease. It comes on in attacks, especially at night and in the early morning, and is worse when the patients lie down. It often begins while the patient is apparently in good health, but is a sure premonition of serious disease.

In some cases these attacks of dyspnœa can be controlled and the patient kept apparently well for months and even years. But as the attacks are repeated, they are more severe and more stubborn. The heart's action fails in addition to the contraction of the arteries, and the dyspnœa becomes of such a character that it can only be relieved by death.

In other patients the first attack of dyspnœa is also the last. It cannot be relieved by any treatment, and continues up to the time of the patient's death.

A catarrhal bronchitis with cough and expectoration is sometimes an annoying symptom. The cough fatigues the patient and it is difficult to control it.

Loss of appetite, nausea and vomiting are frequent symptoms. When they do not already exist it is easy to cause them by the use of improper drugs.

The heart is very often affected. The disease of the kidneys after a time produces hypertrophy of the left ventricle. This does no harm until the time comes when the heart's action fails in spite of the hypertrophy, then the dyspnœa and the dropsy follow.

Chronic endocarditis, chronic myocarditis, and dilatation of the ventricles are associated with chronic nephritis in two ways: They may cause a chronic congestion or degeneration of the kidney, which is afterward followed by a nephritis; or the heart disease and kidney disease are developed in the same person, neither one of them secondary to the other. In these patients it is by no means easy to tell how much of the dropsy, the dyspnœa, and the loss of nutrition belongs to the heart disease and how much to the nephritis.

COURSE OF THE DISEASE.

There is hardly any limit to the variations of the disease, but the most constant symptoms are anæmia, dropsy, and albumen in the urine.

The following are some of the ordinary examples of this form of nephritis:

1. There are cases in which nearly every day for many months small quantities of albumen and a few casts are to be found in the urine. The quantity of the urine, its specific gravity, and the excretion of urea remain normal. There are no changes in the heart or arteries, or in the tension of the arteries, nor are headache or dyspnœa present. The patients may have disturbances of digestion, but no great loss of nutrition unless they have been put on a restricted diet. The condition seems to be a true nephritis and not an albuminuria, yet the prognosis is often good. I have followed some of these patients for years, and seen them continue to be in good health.

This set of mild cases is not as well known as it should be. Some of the cases are supposed to be examples of acute nephritis, and all of them are apt to be over-treated to such an extent as to make them needlessly ill. They do best on a varied diet, with plenty of fresh air and exercise and no drugs, except such as are needed to correct errors of digestion.

2. There are cases in which the symptoms are nearly continuous. The patients begin with anæmia, headache, disturbances of digestion and a little dropsy. No one of these symptoms is at first very marked; the patient is not in bed, he does not feel very sick, but even at this time the urine contains a large quantity of albumen. As the weeks and months go on all the symptoms grow worse: the anæmia is more profound, the headaches and disturbances of digestion more troublesome, the dropsy involves more of the subcutaneous tissue and the serous cavities, so that the patient becomes more and more helpless. There may be intercurrent attacks of acute uræmia with contraction of the arteries, or loss of eyesight, or a troublesome bronchitis. The ordinary duration of this form of the disease is from one to three years. The

patients die with the most extreme dropsy, or in the state of chronic uræmia, or both these conditions exist together.

3. There are cases in which the anæmia, the dropsy, and the dyspnœa come on in attacks which last for weeks or months. Between the attacks the patients are comparatively well, often able to work, although the urine always contains albumen. These patients often go on for a number of years, better in the summer and worse in the winter. But each successive attack is more serious than the preceding, and finally there comes an attack from which the patient does not recover.

4. There are cases in which a number of years before death the patients have an attack of acute or sub-acute nephritis with anæmia, dyspnœa, dropsy, albumen in the urine, and all the usual symptoms. From this they apparently recover completely and seem to be in their ordinary health. The urine continues to contain a little albumen, or on some days the albumen disappears altogether. From year to year the specific gravity slowly falls. The exudation from the blood vessels of the kidneys stops, but the chronic productive inflammation of the kidneys continues. Finally, after exposure, with an accident, with a pneumonia, or without discoverable cause, all the symptoms of a sub-acute nephritis are rather suddenly developed, and the patient soon dies.

5. There are cases which for years have no symptoms except pallor of the skin and mucous membranes, and urine of low specific gravity which habitually contains a moderate quantity of albumen. These patients must not be confounded with cases of simple anæmia, nor with those of persistent albuminuria without kidney disease. The diagnosis is sometimes quite difficult. The prognosis in these patients depends upon the specific gravity of the urine. If it is constantly below 1010 the prognosis is bad, no matter how well the patients may feel.

6. There are cases in which the first symptoms is the attack of spasmodic dyspnœa. This may continue and other renal symptoms rapidly develop. More frequently, however, the dyspnœa is palliated or relieved by treatment. The patient then goes on for months or years with occasional attacks of dyspnœa, each one

more severe and harder to control, until finally other renal symptoms appear.

7. There are the cases complicated with endocarditis, myocarditis, or dilated heart. In these patients we have the association of cardiac and renal symptoms, either one predominating.

8. There are cases in which all the symptoms disappear, and the urine returns to its natural condition. If this improvement continues for a number of years it seems probable that the nephritis has come to a standstill, and that enough kidney tissue has been left unimpaired to carry on the functions of the organ.

9. The hospital cases of this form of nephritis present the characteristic symptoms—the dropsy, dyspnœa, vomiting, diarrhœa, albuminuria, etc., in a marked degree. Patients of this class do not come into the hospital except during an exacerbation of the nephritis, or after it is far advanced. The milder forms of such a nephritis, its earlier stages, its behavior between the exacerbations have to be learned from patients in private practice. For example I have the records of 225 cases of chronic nephritis with exudation treated in the Roosevelt Hospital during a period of eight years.

In all of these patients albumen was present in the urine in large quantities, but yet the variations in quantity on different days were often considerable. In the patients who improved the quantity of albumen and the dropsy usually rose and fell together. The smaller quantities of albumen belonged to the patients in whom the nephritis had lasted for the longest time.

Often in these patients there was a marked discrepancy between the history and the real duration of the nephritis, so that the cases could be mistaken for cases of acute nephritis. Thus a man 32 years old asserted that he was perfectly well and working up to twenty days before his death. He died with dropsy and anæmia. The kidneys were of the large, white variety, far advanced in chronic nephritis.

There is also something deceptive about the improvement of these cases. Patients come into the hospital with much dropsy and marked albuminuria. They go out of the hospital in two weeks with no dropsy and no albumen in the urine, but they are not cured.

There are also cases in which the exacerbation of the nephritis is marked, not by an increase of albumen, but by red and white blood cells in the urine.

Well-marked dropsy was present in 218 of the cases, and in 124 of the cases it was stated that this was the first symptom noted by the patient. In 66 of the cases dyspnœa was the first symptom; in 14 cases headache; and in 12 vomiting. In the cases in which the blood was examined for secondary anæmia the hæmoglobin was from 20 to 80 per cent.

Nine of the patients had attacks of acute uræmia while in the hospital; 41 patients were in the condition of chronic uræmia. Most of these died while in the hospital, but a few were removed by their friends before death.

In 70 cases there was hypertrophy of the left ventricle of the heart, and in 39 cases there was chronic endocarditis.

Fifty-nine of the patients died while in the hospital, and on 37 of these autopsies were made. There were 18 large, white kidneys; 5 small, white kidneys; 5 medium-sized kidneys, with adherent capsules, thick cortices and roughened surfaces; 6 small, nodular atrophied kidneys; 1 large congested kidney; 1 kidney of normal weight, thin cortex, and adherent capsule; and 1 chronic pyelo-nephritis.

Treatment.—We must acknowledge at once our ignorance of the nature of the poison or poisons which produce chronic nephritis with exudation, and also of any direct means of stopping the inflammatory process. The lesson which experience teaches us, a lesson often difficult to learn, is that the whole management and treatment of these cases must be entirely different, according to the severity of the nephritis, the active or passive character of the nephritis at different times, and the presence or absence of certain symptoms.

In the mild cases with but few symptoms, except the albumen and casts in the urine, it would seem at first sight that the nephritis was in an early stage and that energetic treatment might cure it. It is found, however, that these patients do best with very little treatment.

The diet is to be restricted only so far as to exclude unwholesome food. No medicine is required except to correct errors of digestion. Some of the patients can be allowed to continue at their ordinary pursuits. Others are benefited by travel and change of climate.

In the same way the advanced cases in which the nephritis is running a very slow course with but few symptoms must be quietly managed. The digestion, the general nutrition and the secondary anæmia are the features which call for attention.

On the other hand, a more active nephritis, or a period of exacerbation, requires more energetic treatment.

The conditions calling for treatment are:
The nephritis.
The albuminuria,
The dropsy.
The headache and sleeplessness.
The nausea and vomiting.
The contraction of the arteries.
The anæmia.
The dyspnœa.

For the *nephritis* the patient should be kept in bed and placed on a fluid diet. He should have a hot pack twice a day, once a day or every other day, as he will bear it. The most useful drugs are: morphine in very small doses, digitalin gr. $\frac{1}{100}$ and aconite M 1-3.

The quantity of *albumen* in the urine can be diminshed by the hot pack and by the use of digitalin gr. $\frac{1}{100}$ four times a day.

The *dropsy* is favorably affected by the hot pack. It can be sensibly diminished by cathartics, of which elaterium is perhaps the best. But the effect of cathartics is temporary, and their use cannot often be repeated.

For many patients digitalis in some form is the most useful drug for the dropsy, and it can be continued without injury for weeks and months. Favorable results are reported from the use of lactate of strontium in 30-grain doses four times a day, and from diuretin in 15-grain doses three times a day. In some patients caffeine, convallaria, or strophanthus will answer better than digitalis.

If there is much fluid in the serous cavities it should be drawn off with the aspirator. If the dropsy of the subcutaneous connective tissue is excessive the skin is to be pricked and the fluid allowed to drain off.

The *headache* and *sleeplessness* may exist with or without contraction of the arteries. If the arteries are not contracted, morphine in very small doses, codeine, or the bromides can be used. If the arteries are contracted morphine in larger doses and chloral are the best drugs.

The *nausea* and *vomiting* are controlled by the milk diet, or it may be necessary to add an alkali to the milk, or to use peptonized milk or kumyss.

Contraction of the Arteries.—The character of the radial pulse and the heart's action are to be constantly watched. We do not wait for the dyspnœa, or vomiting, or convulsions to make their appearance, but as soon as the pulse shows an increased tension we begin with nitro-glycerin, chloral hydrate, morphine, or potassium iodide. It is to be remembered that morphine in considerable doses can only be given to patients with this form of nephritis when the arteries are contracted. At all other times it is very easy to have dangerous and even fatal results with any preparation of opium.

The *anæmia* of chronic nephritis does not behave like simple anæmia. Iron is not a specific for it, although it may be of service. The greatest improvement in the anæmia is effected by the subsidence of the nephritis.

The *dyspnœa* is dependent either upon contraction of the arteries or upon dropsy. The treatment for it, therefore, is either the treatment of dropsy or the treatment of contraction of the arteries.

In these cases treatment carried on in these ways is often very satisfactory. All the symptoms subside and disappear. But it must be remembered that the kidneys have become changed in their structure, that a chronic productive nephritis still continues, that relapses and exacerbations are to be expected.

As the symptoms subside the patient gets back to a solid diet, is out of bed, and then out of doors. If it is possible for him to spend the next two years in a warm climate where he can lead an out-of-doors life, the probabilities of permanent improvement will be much greater.

In the cases in which the symptoms continue and get steadily worse, treatment is very unsatisfactory. We try the measures that have just been described, but they are of no avail. We cannot even alleviate symptoms, the drugs do not help at all. After a time it becomes evident that there is no use in continuing plans of treatment which do nothing, and we employ very little treatment.

In the cases with chronic endocarditis, myocarditis, or dilatation of the ventricles, the management of the heart's action becomes a matter of great importance.

CASE XXIX. *Sub-Acute Productive Nephritis, Followed Nine Years Later by Chronic Nephritis.*—Male, 25 years old, December 30, 1884. When he was 16 years old he had an attack of vomiting, with dropsy of the legs and scrotum.

For the past two weeks he has had vomiting, dropsy of the legs and scrotum; urine 1.008, a large quantity of albumen and casts. He improved in the hospital and was discharged in fair condition. June 30, 1886, he was readmitted. He has been doing badly for the past month, with headache, nephritis, retinitis, dyspnœa, hard radial pulse and daily vomiting. Urine 1.020, urea 4 grains to the ounce, a good deal of albumen, no casts.

July 12, 1886, he was discharged somewhat improved.

July 30 he was re-admitted with the same symptoms and discharged August 12.

August 25 he was re-admitted with the same symptoms, but the dyspnœa was now constant and urgent. He continued to get worse in the same way, dropsy supervened, and he died October 12, 1886.

Autopsy.—The legs are œdematous. There is serum in both pleural cavities. The pericardium is coated with fibrin, the aortic valves are a little thickened, both ventricles are moderately dilated and hypertrophied.

The liver and stomach are congested; the spleen is normal.

The kidneys are small, their capsules adherent, their surfaces nodular, the cortex mottled red and gray. There is an extensive diffuse growth of connective tissue in the stroma. A large number of the cortex tubes are either compressed or dilated. In the glomeruli there is a marked growth of tuft cells, in some of them atrophy of the capillary tuft; the arteries are but little thickened.

CASE XXX. *Chronic Nephritis, with Exudation of About Four Years' Duration; Dyspnœa the First Symptom.*—Male, 42 years old, March 25, 1887. For two years he has been troubled with dyspnœa on exertion and occasional headache. One week ago the dyspnœa became worse and he developed general anasarca. Now he is anæmic, with general dropsy, headache, temperature 98° to 101°, pulse 96, with increased tension; urine 1.012, with a large quantity of albumen.

He improved in the hospital and was discharged May 3, 1887, in fair condition. April 12, 1889, he was re-admitted.

Now he is emaciated, anæmic, pulse 164, breathing 32, temperature 102°, with coarse râles over both lungs. Urine 1.014, contains large quantities of albumen, many casts and red and white blood cells. He had several convulsions followed by delirium, and died on April 14.

Autopsy.—The dura mater is adherent to the skull, there is an increase of serum beneath the pia mater, the arteries at the base show a marked thickening of the muscular coat.

A large part of the right lung is in the condition of incomplete red hepatization. The left ventricle of the heart is hypertrophied, there is old thickening of the aortic and mitral valves.

The liver is large and fatty, the spleen is normal.

The mucous membrane of the stomach is thickly coated with mucus.

The kidneys are large, their capsules adherent, their surfaces coarsely nodular, the cortex thick, gray and white. The cortex tubes show marked swelling and degeneration of the epithelium, there are large sub-capsular wedges of connective tissue containing atrophied tubes and glomeruli, the other glomeruli are large with swollen tuft cells, the arteries are very little changed.

CASE XXXI. *Chronic Nephritis, with Exudation; Ordinary Hospital Case.*—Male, 29 years old, November 17, 1889. He is intemperate, but denies venereal disease. For about two months the urine has been passed more frequently, there has been dyspnœa and dropsy of the feet and legs. These symptoms have not been continuous.

Two weeks ago the feet and legs and the scrotum, pelvis and face became swollen, there was dyspnœa on exertion, a little cough, no headache, no vomiting, the appetite remained good. He complains chiefly of the dropsy.

Now he is pale, with moderate general dropsy, tongue clean, appetite good, sleeps well, pulse 54, no tension, breathing 22, temperature 98.4°. Urine 1.013, 40 ounces; albumen, large quantity; granular and epithelial casts; urea 6 grains to the ounce.

Hæmoglobin 80 per cent.

November 20 the dropsy has gone, but the quantity of albumen in the urine remains the same.

December 1 he is out of bed and doing well, but the quantity of albumen in the urine remains the same.

December 6 he is beginning to have severe headaches.

December 10 he had two general convulsions; pulse 125, with increased tension.

December 21 he is out of bed, feeling well; no tension of the pulse.

December 25, headache again; temperature 99.6°.

December 28, headache, vomiting, temperature 101.8°.

December 29, pain in the chest, vomiting; temperature 102.8° to 104.6°.

January 2, temperature 103° to 102.6°; physical signs of consolidation of the right lung.

The urine throughout was passed in fair quantity, contained a good deal of albumen with hyaline and granular casts.

January 3 he died.

Autopsy.—The brain was not examined. The body is emaciated, with very little dropsy.

There is purulent serum in the right pleural cavity. The upper lobe of the right lung is hepatized.

The mitral valve is thickened and insufficient; the walls and cavities of the heart are normal.

The liver is fatty, the spleen is large. The kidneys weigh together 24 ounces; their surfaces are smooth, the cortex thick and yellow. There is an extensive diffuse growth of connective tissue in the stroma. In the tubes there is marked granular and fatty degeneration of the epithelium. The glomeruli are large, with a marked growth of the tuft cells. The walls of the arteries are a little thickened.

CASE XXXII. *Chronic Nephritis, with Exudation; Chronic Uræmia.*—Male, 26 years old, February 22, 1889. Two years ago he had dropsy of the feet and this has since been repeated several times. He has lost strength, his memory has been poor, he has had several attacks of unconsciousness. All these symptoms have become more marked; for the past two weeks he has been drowsy or irritable. Now his tongue is dry and brown, no appetite, pulse 86, breathing 16, temperature 98°. He lies quietly in bed, taking no notice of anything, but can be roused and answers questions intelligently. Urine 1.014, contains much albumen and many casts; urea 6 grains to the ounce.

He became gradually more stupid, with muscular twitchings, had one general convulsion and died February 26.

Autopsy.—The large arteries at the base of the brain are normal; there are little aggregations of small cells scattered through the pia mater.

The lungs are congested and œdematous, with patches of broncho-pneumonia in the lower lobes.

The left ventricle of the heart is hypertrophied; the valves are normal.

The liver and spleen are normal. The mucous membrane of the stomach and of the upper part of the small intestine is black and coated with mucus.

The kidneys are small, their capsules adherent, their surfaces finely nodular, the cortex thin and white. There is an extensive diffuse growth of connective tissue in the stroma. The tubes are compressed and deformed. In the glomeruli there is a marked growth of tuft and capsule cells, with obliteration of many of the capillary tufts. All the coats of the arteries are much thickened.

CASE XXXIII. *Chronic Nephritis, with Exudation of Five Years' Duration; Repeated Attacks of Headache.*—Male, 28 years old, December 14, 1884. For five years he has had attacks of frontal headache, nausea and vomiting coming on in the morning and lasting all day. These attacks were repeated every week up to two weeks ago; since then they have occurred every other day.

Now the man is fairly nourished; pulse 99, temperature 100°, urine 1.008, contains much albumen, many casts and red and white blood cells.

December 16 he became drowsy and stupy, with subcutaneous dropsy, which increased.

December 18 he died comatose.

Autopsy.—There is general subcutaneous œdema. The lungs are congested and œdematous, with commencing red hepatization of the lower lobes.

The left ventricle of the heart is hypertrophied, the valves are normal. The liver is congested, the spleen is a little enlarged, the stomach and intestines are normal. The kidneys are large, the cortex thick, mottled red, gray and white. There is an extensive growth of connective tissue in the stroma. The tubes are compressed, their epithelium flattened. In the glomeruli there is a marked growth of tuft cells. The arteries are normal.

CASE XXXIV. *Chronic Nephritis, with Exudation; Acute Symptoms Brought on by Pneumococcus Infection.*—Male, 40 years old, April 1, 1889. He was found comatose in his room, was brought into the hospital and had several convulsions during the day. Pulse 142, breathing 22, temperature 101°, urine 1.017, large quantity of albumen and blood.

He continued in the condition of alternating delirium and coma until he died on April 6.

Autopsy.—The brain was not examined. The right lung is congested, œdematous and in the condition of commencing red hepatization.

There are old pericardial adhesions and some recent fibrin. The left ventricle is hypertrophied, there is a little old thickening of the aortic and mitral valves, the arch of the aorta is atheromatous. The liver is large and congested, the spleen is normal.

The kidneys are large and congested, their capsules adherent, their surfaces nodular. The epithelium of the cortex tubes is swollen and granular, many of the tubes contain blood. There are large sub-capsular wedges of connective tissue containing atrophied tubes and glomeruli. The tuft cells of the glomeruli are swollen.

CHRONIC PRODUCTIVE NEPHRITIS WITHOUT EXUDATION.

Definition.—A chronic inflammation of the kidney attended with a new growth of connective tissue in the stroma, permanent changes in the glomeruli, degeneration of the renal epithelium, and sometimes changes in the walls of the arteries.

Synonyms.—Chronic Bright's disease; Cirrhosis of the kidney; Granular degeneration; Interstitial nephritis; Chronic indurative nephritis; The arterio-sclerotic kidney.

Etiology.—While this form of nephritis is especially common in persons over forty-five years old, it is by no means rare in young adults, and is occasionally seen in children.

It seems to be caused by chronic alcoholism, lead poisoning, gout, and by the same conditions as those which cause emphysema, endocarditis, and cirrhosis of the liver. It follows chronic congestion of the kidney, hydro-nephrosis, and chronic pyelitis.

Morbid Anatomy.—The larger number of the affected organs are found after death to be diminished in size; the two kidneys together may not weigh more than two ounces. The capsules are adherent; the surfaces of the kidneys are roughened or nodular, the cortex is thin and of a red or gray color.

A considerable number of these kidneys, however, do not differ in their size or appearance from normal kidneys, except that their capsules are adherent and their surfaces roughened.

Occasionally the kidneys are large, weighing together from 16 to 32 ounces, with smooth or nodular surfaces, and a cortex of red, gray or white color.

If the nephritis follows chronic congestion the kidneys remain hard, but the cortex becomes thinned, the capsules adherent, and the surface roughened.

There is a growth of new connective tissue in the cortex and also in the pyramids, which becomes more and more extensive as the disease goes on. In the cortex the new tissue follows the distribution of the normal sub-capsular areas of connective tissue, is in the form of irregular masses, or is distributed diffusely between the tubes. In the pyramids the growth of new connective tissue is diffuse.

The tubes, both in the cortex and pyramids, undergo marked changes. Those included in the masses of connective tissue are diminished in size, their epithelium is flattened, some contain cast matter, many are obliterated. The tubes between the masses of new connective tissue are more or less dilated; their epithelium is flattened, cuboidal, swollen, degenerated, or fatty. The dilatation of the tubes may reach such a point as to form cysts of some size, which contain fluid or coagulated matter. These cysts follow the lines of systems of tubes, or are situated near the capsules.

Of the *glomeruli* a certain number remain of normal size, but with the tuft cells swollen or multiplied. Many others are found in all stages of atrophy and of change into connective tissue. The atrophy seems to depend partly on the growth of tuft cells and intra-capillarly cells, partly on the thickening of the capsules, partly on the occlusion of the arteries. If the chronic nephritis follows chronic congestion of the kidneys the glomeruli remain large, with an increased growth of tuft cells, or they become atrophied, but with the dilatation of the capillaries still evident. The capillaries of the glomeruli may be the seat of waxy degeneration. The arteries exhibit the same changes as have already been described in speaking of chronic exudative nephritis.

Complicating Lesions.

Heart.—Hypertrophy of the left ventricle of the heart is frequently caused by exudative nephritis, but much more frequently by chronic nephritis without exudation. It must be admitted, however, that such an hypertrophy, although frequent, is not constant; that with both exudative and non-exudative nephritis there may be no change in the wall of the left ventricle. The hypertrophy of the wall of the ventricle may, after a time, be succeeded by dilatation or chronic degeneration, or myocarditis.

Chronic endocarditis is often associated with this form of nephritis, apparently both lesions being produced by the same cases. It may also happen that chronic endocarditis causes first chronic congestion of the kidneys and then chronic nephritis without exudation.

Lungs.—Pulmonary emphysema and cirrhosis of the liver are frequently associated with chronic nephritis.

A number of statistics have been given as to the relative frequency of some of the associated lesions of chronic nephritis.

I will quote three sets of cases:

Southey (Lumleian Lectures, 1881) gives the results of 550 autopsies made at St. Bartholomew's Hospital; of these 358 were granular kidneys, 141 were white or mottled kidneys, 35 were amyloid kidneys, 16 were acute scarlatinal kidneys.

Bamberger (Volk. Klin. Vorte, No. 173) reports 2,430 autopsies of Bright's disease made during twelve years at Vienna. Of these 807 were primary, 1,623 were secondary. He separates the cases into those of acute Bright's disease, those of chronic Bright's disease, and those of atrophied kidneys.

My own records are from autopsies made at Bellevue and Roosevelt Hospitals on 1,087 patients in whom well-marked chronic nephritis was found after death.

	LARGE WHITE KIDNEYS.			ATROPHIED KIDNEYS.		
	Southey.	Bamberger.	Delafield.	Southey.	Bamberger.	Delafield.
Whole No....	141	....	398	358	....	689
Endocarditis.	27.7%	....	44%	26.7%	....	56%
Hypertrophy of L. Ventricle......	19%	34.2%	23%	67%	54%	36%
Apoplexy....	....	8%	5 cases	22%	13.6%	0.87%
Cirrhosis.....	....	....	19.8%	0.55%	....	16%
Emphysema .	....	....	14%	....	....	20%

Arteries.—One of the most important of the complicating lesions is chronic arteritis. The relationship between arteritis and nephritis, and the ways in which they are associated together, are not as fully understood as they should be. The principal reason for this is the failure to recognize the fact that chronic inflammation of the walls of the arteries is just as much a disease as chronic endocarditis, or emphysema, or cirrhosis of the liver.

Unquestionably arteritis is more often seen associated with other diseases than by itself. It must also be admitted that it is of such frequent occurrence in old persons that it is natural to think of it as a senile change. Still further, the use of such names as arterio-capillary fibrosis and arterio-sclerosis have helped to prevent us from classing arteritis with the other chronic productive inflammations.

Chronic inflammation may involve the entire aortic system of arteries, or it may be confined to a part of that system.

If only the arteries in some one part of the body are involved, then in that part of the body the blood supply is irregular or cut off, the diseased artery may become dilated, or it may rupture.

If a large part of the aortic system of arteries is involved, then the patients suffer from symptoms which seem to depend partly upon the changes in the arteries, partly upon attacks of contraction of the arteries, partly upon hypertrophy of the left ventricle of the heart and heart failure, partly upon the obstruction to the passage of blood through the cerebral arteries.

The clearest idea of general arteritis as a disease is to be obtained by observing it in persons not over forty years old, who have no complicating lesions.

At first for a number of years these patients only suffer from impaired nutrition, a disposition to become anæmic, and attacks of dyspnœa. It can be seen and felt that the walls of the temporal and radial arteries are thickened and that the left ventricle of the heart is hypertrophied. At the times when the patient has dyspnœa the tension of the pulse is much increased.

For a considerable length of time the nutrition and the anæmia can be improved by climate and by diet. The attacks of dyspnœa can be controlled by the drugs which dilate the arteries. But sooner or later the patients get worse. Some of them get up a dyspnœa that cannot be controlled, the action of the hypertrophied heart fails, and the patients, after suffering for weeks or months with the most distressing symptoms, die. In other cases death takes place with cerebral symptoms—sudden unconsciousness, or aphasia, or hemiplegia. After the death of these patients no lesions of any consequence are found except the changes produced by chronic inflammation of the walls of the arteries.

It is evident that the symptoms and death of these patients are due to the changes in the arteries; that the disease from which they have suffered is chronic arteritis. But it is also evident that their symptoms—loss of nutrition, anæmia, contraction of the arteries, hypertrophy of the left ventricle, dyspnœa, heart failure, unconsciousness, aphasia, hemiplegia—are also the symptoms of chronic nephritis.

Still further we find that many patients with these symptoms do have both arteritis and nephritis. In any given case with these symptoms, therefore, it is a matter of importance to determine whether the patient has arteritis alone, or nephritis alone, or both diseases at the same time.

Patients who have chronic nephritis are more liable than are other persons to attacks of pericarditis, bronchitis and gastric catarrh.

Symptoms.—The Urine.—The typical urine of chronic non-exudative nephritis is a urine increased in quantity, of a specific gravity of about 1.010, containing a diminished quantity of urea, without albumen or casts, or with a trace of albumen and a very few casts. But exacerbations of the nephritis and changes in the circulation may for a time considerably increase the quantity of albumen and the number of casts.

Very important modifications of the urine, however, are of ordinary occurrence. It is quite possible, with nephritis of this type far advanced, to have urine not below 1.023 in specific gravity and without albumen or casts. When one sees this urine during life and then the kidneys after death it is difficult to understand how they can belong to each other.

On the other hand, there are cases in which the specific gravity of the urine falls almost to 1.000, either with or without waxy degeneration of the blood vessels. In some cases the quantity of urine is very much increased—several quarts in the twenty-four hours. During the attacks of contraction of the arteries, to which these patients are liable, the urine may be diminished to a few ounces or even suppressed.

Cerebral Symptoms.—Of the patients who die from apoplexy or embolism of the cerebral arteries it is found that a considerable number have chronic nephritis.

It is also found that a large number of patients with chronic nephritis have cerebral symptoms which seem to depend partly on the presence of poisons in the blood, partly on the condition of the arteries and heart, and partly on contraction of the arteries. These cerebral symptoms belong to an advanced nephritis, but yet they may be one of the first symptoms to show that such a nephritis exists.

Headache and sleeplessness are often present, the headache sometimes so severe and continuous that the patient is nearly maniacal. Instead of the headache there may be neuralgic pains in different parts of the body.

Muscular twitchings and general convulsions are much more serious. They may be early symptoms, or not occur until late in the disease.

Hemiplegia, with or without aphasia, may be the first symptom to call attention to the nephritis, or may not occur until later in the disease. The invasion of the hemiplegia is sudden and is usually accompanied by coma. There is loss of motion alone, or of both motion and sensation. The hemiplegia, aphasia, and coma may continue up to the time of the patient's death, or disappear after a few hours or days. In the latter case the patient may have several such attacks. These attacks have been ascribed to localized œdema of the brain. In the cases which I have seen there were no changes in the brain tissue, but the cerebral arteries were damaged by chronic arteritis.

Delirium, mild or violent, stupor, and coma may come on in sudden attacks, or be developed slowly and gradually.

When these cerebral symptoms come on in attacks the pulse is of high tension, the temperature is raised, and the patients are said to suffer from acute uræmia. Very often they recover from a number of these attacks. In the fatal attacks the pulse often loses its tension and becomes rapid and feeble; the patients die comatose with a feeble heart.

Instead of such acute attacks of cerebral symptoms, delirium and stupor may come on gradually in persons far advanced in their nephritis. The temperature is then apt to be below the normal and the pulse is rapid and feeble.

Temporary blindness, neuro-retinitis, or nephritic retinitis are developed in a moderate number of patients.

Chronic bronchitis and emphysema very frequently exist and their symptoms often form a large part of the clinical history.

Attention has already been called to the large share that chronic arteritis may have in the production of some of the renal symptoms.

The Heart.—The left ventricle of the heart regularly becomes hypertrophied after the nephritis has lasted for several months. The disposition to hypertrophy is, I think, rendered greater by repeated attacks of contraction of the arteries and by complicating arteritis. The hypertrophy is usually easily made out. The patient remains unconscious of its existence, or has disturbances of sensation and palpitation. As the disease goes on the hypertrophied heart may become feeble, and then dyspnœa and the other evidences of feeble circulation make their appearance.

In the same way the complicating endocarditis, which so often exists, may give no trouble until the valves are a good deal changed, or the ventricles dilated, or the heart's action altered, or the arteries contracted; then the circulation is interfered with, and the results of venous congestion of different parts of the body show themselves.

Dyspnœa is a frequent symptom, often the first symptom noticed by the patient. It is a spasmodic dyspnœa, coming on in attacks which last for minutes, hours, or days. It is made worse by bodily or mental exertion, or by the recumbent position. It does not resemble bronchial asthma. It is apparently due to the association of changes in the arteries and heart. It cannot be distinguished from the dyspnœa which is caused by arteritis without nephritis. With contraction of the arteries alone, or with a feeble heart alone, no dyspnœa may exist; but if the contraction of the arteries be so great that the hypertrophied heart cannot overcome the obstruction, or if with contraction of the arteries the heart becomes dilated or feeble, then the attacks of dyspnœa begin. At first the attacks are not severe and are of short duration, but if the mechanical conditions which cause them cannot be controlled, they become longer and more distressing.

The stomach may continue to perform its functions fairly well, but more often there is gastric indigestion, gastric catarrh, or spasmodic vomiting.

Dropsy does not seem properly to belong to chronic nephritis without exudation. When it is present there will usually be found to account for it either endocarditis, dilatation of the ventricles, fatty heart, a feeble heart, pulmonary emphysema, or cirrhosis of the liver.

Profuse bleeding from the pelvis of atrophied kidneys is sometimes seen. In all cases, after a time, the nephritis exerts its effects upon the nutrition of the patient, and the flesh and strength are diminished. On the other hand, the patients do not usually become as pale as they do with an exudative nephritis.

Course of the Disease.—It is characteristic of the chronic productive inflammations of the lungs, the heart, the arteries, the liver, and the kidneys, that, while they often exist as serious and fatal diseases, they may also exist as lesions and yet do not interfere with long life and apparent good health. This seems to depend, at least in part, on the rapidity with which the inflammatory changes in these different parts of the body are developed. If the development is slow enough, the functions of the organ continue to be performed in spite of the new growth of connective tissue.

We have to admit that in all cases of chronic non-exudative nephritis a period of weeks, or months, or years elapses during which the changes in the kidneys are slowly going on, and yet the patients seem well and are not aware that they have any disease. How far the nephritis can advance and how many years it can exist before the symptoms of it appear, it is difficult to say. We see a great many different stages in the development of the nephritis in persons who die from other diseases and have never given any renal symptoms.

The nephritis is of slow development, gradually altering the structure of the kidney more and more, so that we should expect that the symptoms of the nephritis would also be developed gradually. This is very often the case, but quite as often the nephritis will advance without symptoms up to a certain point and then the patient suddenly becomes ill.

A. Cases with Slow Development of Symptoms.—Of the patients in whom the symptoms are gradually developed we may distinguish:

1. Patients who gradually develop hypertrophy of the left ventricle of the heart, with a lowering of the specific gravity of the

urine, and a pulse that is easily made too tense; otherwise their health is good. We often watch these persons for many years, expecting other renal symptoms. But the symptoms do not come, and the patients die of some other disease.

2. Patients who have digestive disturbances, and gradual loss of flesh and strength. The urine is of low specific gravity and increased quantity, or the specific gravity and quantity remain almost normal; often from time to time there are traces of albumen and a few hyaline casts. These patients are often very puzzling. From year to year they slowly get more feeble and more emaciated; the digestive disturbances are sometimes better and sometimes worse. Occasionally there is an interval of great improvement, so that the patients think they have entirely recovered. As the disease lasts a long time the patients are apt to see a number of physicians and get a number of opinions, for the diagnosis is really a difficult one. Some of the patients die from intercurrent diseases, but others go on and die, simply exhausted with nothing but the chronic nephritis.

3. Patients who for months or years have attacks of spasmodic dyspnœa, and between these attacks are comparatively well. The patients are usually over forty years of age. The attacks of dyspnœa are apt to come on in the early morning and go off later in the day. Often chronic arteritis, or chronic endocarditis, exists at the same time. For a while the attacks of dyspnœa can be relieved, and the patients are capable of mental and physical exertion and feel quite confident of recovery. But as the attacks of dyspnœa recur they last longer and are harder to relieve. Finally comes the time when the dyspnœa cannot be relieved. It lasts day and night, the patients cannot lie down, the scrotum and legs become œdematous, and death hardly comes soon enough to relieve their distress.

4. Patients who have symptoms progressing for several years. At first vomiting, or headache, or neuralgic pains. Then dyspnœa, a little dropsy of the legs, and loss of flesh and strength. Finally death from exhaustion, or with an attack of convulsions, or in coma.

5. Patients in whom the symptoms come on in attacks, each

attack worse than the preceding, and the general health more and more impaired between the attacks. During the attacks there are headache, sleeplessness, delirium, stupor, coma, convulsions, dyspnœa, vomiting—sometimes one, sometimes another the prominent symptoms. The tension of the pulse is considerably increased. The urine is of low specific gravity and often contains a little albumen. Between the attacks the patients at first seem to be fairly well, but later they gradually lose flesh and strength. The urine between the attacks is of low specific gravity and contains little or no albumen. The patients finally die in one of the attacks.

B. Cases with Rapid Development of Symptoms.—Of the patients who are apparently in their ordinary health until there is a violent invasion of symptoms, we may distinguish those in whom the attack seems to be precipitated by an injury or an intercurrent disease, and those in whom it comes on without discoverable cause. In either case the attack regularly takes the form of cerebral symptoms, or of dyspnœa, or of vomiting, or of sudden death. During these attacks the tension of the pulse is high, the urine is diminished in quantity or suppressed, and often contains a little albumen.

1. The cerebral symptoms are: general convulsions, coma, hemiplegia, and aphasia. The convulsions come on suddenly, they are repeated several times, between them the patients are unconscious. Many of the patients die with the convulsions, but a few recover. We are apt to see these persons for the first time while the convulsions are going on, and are told by their friends that they were in their ordinary health until the convulsions began.

The coma is developed in the same rapid way. The patient is found in bed, in a room, or in the street, at first stupid and muttering incoherently, then completely comatose. From this coma they do not emerge, but go on and die in a few hours or days.

The hemiplegia is like that with a clot or with an obstructed artery. The patient falls to the ground unconscious and hemiplegic. If the hemiplegia is on the right side there is usually aphasia. The paralyzed side of the body may remain quiet, or become rigid, or be moved involuntarily. The hemiplegia and unconsciousness usually continue up to death. But occasionally we see a patient who recovers both motion and consciousness.

2. The dyspnœa often starts with an ordinary bronchitis. The patients cannot lie down, they suffer from the constant feeling of dyspnœa, the pulse is full and tense, the drugs which usually dilate the arteries are of little or no effect, the scrotum and legs become œdematous. The patients only live a few weeks.

3. The vomiting may at first resemble that of acute gastritis, or that caused by some irritating substance in the stomach. But it continues, it is exhausting, it does not yield to the ordinary remedies directed to the stomach; the pulse is full and tense. Such vomiting, however, can often be stopped by the drugs which dilate the arteries.

4. The patients, after an injury, or a surgical operation, or without discoverable cause, become feeble, the heart's action is feeble, the urine is diminished or suppressed, and in a few hours the patient is dead. These cases are not common. They are very disagreeable for the physician, as the patients seem to die without sufficient cause.

5. *Hospital Cases.*—The autopsy room of a general hospital affords a large number of examples of chronic nephritis without exudation for anatomical study. But the wards of the same hospital give a much smaller number of cases for clinical study. The reason for this is that so many of these patients come into the hospital for some other disease instead of the nephritis which also exists.

Thus, during a period of eight years at the Roosevelt Hospital, I have records of only 115 well-marked cases of chronic nephritis without exudation. These were all patients who came into the hospital with acute uræmia, with dyspnœa, or for the fatal termination of the disease.

In 77 of the cases albumen was present in the urine while the patients were in the hospital, usually only a trace, but sometimes in considerable quantity.

Dropsy was present in 54 of the cases. In most of these patients chronic endocarditis, dilatation of the ventricles, fatty heart, feeble heart, chronic arteritis, pulmonary emphysema, or cirrhosis of the liver seemed to be largely responsible for the dropsy. But yet I have not included in this set of kidney cases the ordinary marked examples of cardiac and hepatic dropsy.

Thirty-four of the patients had attacks of acute uræmia with cerebral symptoms while in the hospital. In all of these patients there was a marked increase in the tension of the pulse during the attack, and in 20 of them the temperature was over 101° F. during the attack; the highest temperature was 109° F.

Twelve of the patients died in the condition of chronic uræmia.

Chronic endocarditis was present in 21 cases, hypertrophy of the left ventricle of the heart in 57 cases, cerebral apoplexy in 2 cases, cirrhosis of the liver in 8 cases, pulmonary emphysema in 39 cases.

The symptom first noticed by the patient was in 56 cases dyspnœa, in 10 cases gradual loss of flesh and strength, in 6 cases dropsy, in 5 cases vomiting, and in 4 cases headache.

Forty-nine of the patients died of nephritis while in the hospital, and autopsies were made in 45 of these cases. There were 28 atrophied kidneys, 4 medium-sized, hard, granular kidneys; 8 large, hard kidneys; 2 medium-sized smooth kidneys, and 3 large white kidneys.

Treatment.—The progress of the nephritis can be favorably affected by attention to the diet and mode of life, and to climate. As regards the diet, the quantity of sugars and starches taken should be restricted, and the ingestion of fats encouraged. The use of wine, spirits and tobacco should be discontinued. Exercise in the open air is to be advised as long as the strength permits of it. As regards climate, we must consult the idiosyncrasy of the patient; it should be a climate where he eats well, sleeps well and feels well. There is a decided advantage in not remaining in the same place throughout the year.

In the patients belonging to group one, with urine of low specific gravity and hypertrophy of the left ventricle, it will be found that whenever the tension of the pulse is increased the patients do not feel quite so well. When this is the case potassium idodide will often soften the pulse and remove the discomforts.

These patients can also be much improved by regulated exercise in the open air.

In the patients belonging to group two the treatment is directed to the digestive disturbance and the nutrition. The regulation of the diet and the mode of life, lavage of the stomach, relieving constipation, and increasing the production of bile are all of importance. When the production of urine is largely in excess of the normal, combinations of nux vomica and sodium bromide will sometimes act as a specific in reducing this undue quantity.

In the patients with attacks of spasmodic dyspnœa much can be done with the drugs which dilate the arteries and stimulate the heart. According to the tension of the pulse and the strength of the heart's action, we use these drugs separately or together. Chloral hydrate, nitro-glycerin and potassium iodide are the most reliable of the arterial dilators; digitalis, strophanthus and caffeine are the best cardiac stimulants for this purpose.

The treatment of the attacks of headache, convulsions, coma, hemiplegia and vomiting is a matter of importance. The only working theory that one can go on is to believe that at the time of these attacks there is an irritant poison in the blood which causes contraction of the arteries, and that the cerebral symptoms are due partly to the contraction of the arteries and partly to the poison itself. What the poison is or whether it is in all cases the same poison we do not know.

Evidently the indications given for treatment by this theory are, first, to remove the poison from the blood and, second, to dilate the contracted arteries.

The plans which are ordinarily used to remove the poison from the blood are: general blood-letting, purging, sweating, and diuresis. These measures unquestionably can do much good. Whether they do so because they remove poison from the blood, or because they relieve the arterial tensions, is a matter open for discussion.

Dilatation of the arteries can be effected by hypodermic injections of morphine; by nitro-glycerin, chloral hydrate and potassium iodide, and by sweating. Very often with these remedies the pulse will become soft and the cerebral symptoms will disappear. But after the nephritis has advanced beyond a certain point it is found that all these remedies are inert; the tension of the pulse and the cerebral symptoms continue. Or, instead of this, the pulse loses its tension, becomes rapid and feeble, the cerebral symptoms continue and the patients die.

CASE XXXV. *Chronic Nephritis, without Exudation; Dropsy and Uræmia without Albumen in the Urine.*—Male, 30 years old, September 28, 1883. One year ago he first noticed dropsy of his feet and hands, later of his whole body; the urine was scanty. He has had more or less dropsy ever since. Lately he has had cough, with muco-purulent sputum and diarrhœa.

Now he has moderate, general subcutaneous dropsy, pulse 100, with increased tension, breathing 32, temperature 99° Urine for 14 hours 32 ounces, 1.011, no albumen.

September 29, in the morning, he had three general convulsions and died.

Autopsy.—There is general subcutaneous dropsy. The large arteries at the base of the brain are normal. The pia mater is somewhat congested, with old general opacity and an increase of serum. The brain is not anæmic or œdematous.

The left ventricle of the heart is hypertrophied, there is a little old thickening of the aortic and mitral valves.

The lungs are œdematous, the right pleural cavity is two-thirds full of serum.

The liver weighs 4½ pounds and is fatty. The stomach is congested. The spleen and intestines are normal. The kidneys weigh together 12 ounces, the capsules are not adherent, the surfaces are smooth, the cortex is of natural thickness, the kidneys look normal to the naked eye. The changes in the kidneys are not in proportion to the symptoms. There are small patches of connective tissue in the cortex. The capsules of the glomeruli are moderately thickened and the capsule cells are swollen. The epithelium of the convoluted tubes is but little changed.

CASE XXXVI. *Chronic Nephritis, without Exudation; Acute Uræmia, with Temperature of 108°.*—Male, 50 years old, December 16, 1886. He has been intemperate, with morning nausea. For the past year he has had occasional swelling of the legs, headache, loss of strength, pains in the legs and an increased quantity of urine.

Now he is rather fat, has a little œdema of the feet, pulse 116, with high tension, urine 1.010, a little albumen, no casts.

December 19 very stupid, temperature 105° to 108°.

December 20, temperature 100°, died in the afternoon.

Autopsy.—The brain and its membranes are normal, except for a little thickening of the arteries at the base. The lungs are emphysematous. There is marked hypertrophy of the left ventricle of the heart; the mitral valve is thickened and insufficient. There are patches of thickening in the aorta.

The liver is congested, the spleen is normal. The kidneys are small, their capsules adherent, their surfaces nodular, the cortex thin and gray. In the cortex there are large masses of connective tissue enclosing atrophied tubes and glomeruli. In the remaining cortex tubes the epithelium is swollen or flattened. In the glomeruli there is a growth of tuft cells, the walls of the arteries are thickened.

Case XXXVII. *Chronic Nephritis, without Exudation; Loss of Flesh and Strength; Dyspnœa; Sudden Death.*—Female, 56 years old, January 20, 1882. Three months ago she began to have dyspnœa, sleeplessness, scanty urine, loss of flesh and strength and a little swelling of the legs.

Now she is feeble and anæmic, pulse 72, breathing 22, temperature 98°, urine 6 ounces in 24 hours, 1.015, a little albumen.

February 1, 10 days later, she died suddenly.

Autopsy.—The arteries at the base of the brain are tortuous and thickened. The left ventricle of the heart is much hypertrophied, the aortic valves are a little thickened.

Both pleural cavities are half-full of serum.

The liver and spleen are normal. The aorta and large arteries are atheromatous.

The kidneys weigh together 4 ounces. In the cortex there are large masses of connective tissue containing atrophied tubes and glomeruli; between these the cortex tubes are dilated with flattened epithelium. The walls of the arteries are much thickened.

Case XXXVIII. *Chronic Nephritis, without Exudation; Hemiplegia and Coma.*—Female, 50 years old, October 10, 1882. Two weeks ago she suddenly became hemiplegic on the right side with aphasia.

October 8 she began to have involuntary contractions of the right arm and leg. Now she can move all her limbs a little but cannot speak; she is unconscious; the pupils are normal. She continued unconscious, with the right side of the face and the right arm and leg in constant motion, until she died, October 12, two days later.

Autopsy.—The pia mater is a little opaque, but with no increase of serum. The walls of the large arteries are thickened, but none of them are occluded.

The walls of the aorta and of the large arteries are atheromatous.

The left ventricle of the heart is hypertrophied; the walls of the coronary arteries are thickened.

The lungs are congested and œdematous. The liver and spleen are normal. The kidneys weigh together 2 ounces. Both the cortex and pyramids are atrophied, with a large growth of connective tissue in the stroma.

CASE XXXIX. *Chronic Nephritis, without Exudation; Rapid Development of Symptoms.*—Male, 28 years old, May 26, 1884. About three months ago his eyesight began to fail, but otherwise he felt well.

One month ago he had an attack of acute articular rheumatism from which he recovered, but was left with pains over the chest and dyspnœa. He is now emaciated, cyanotic, with marked dyspnœa, pulse 112-128, breathing 24-34, temperature 98°-99°, urine 1.012, some albumen, no casts. There is a loud systolic murmur at the apex transmitted to the left, and a diastolic murmur at the base, transmitted down the sternum. He coughs up mucus streaked with blood. He has a nephritic retinitis; there is no dropsy.

June 1: There were convulsive movements of the muscles of the face and arms; he became unconscious; he died with stertorous breathing and feeble heart on June 2.

Autopsy.—The brain was not examined. The left ventricle of the heart is dilated and hypertrophied, the valves are normal. The lungs are congested and œdematous. The liver and spleen are normal. The stomach and small intestine contain blackish, greenish fluid.

The kidneys are very small, their capsules adherent, their surfaces nodular, the cortex and pyramids atrophied.

PUERPERAL ECLAMPSIA.

During the later months of pregnancy, during labor, and immediately after childbirth, women not infrequently become anæmic and dropsical, have albumen in the urine, and develop alarming cerebral symptoms: headache, blindness, convulsions, and coma.

These symptoms are especially frequent in primiparæ, in young women, and with twin pregnancies. They may be repeated in several successive pregnancies. They belong to the second half of pregnancy, increasing in severity as the pregnancy advances, and are at their worst during labor.

Morbid Anatomy.—In women dying with cerebral symptoms and albuminuria at about the time of childbirth, I have found the kidneys in the following conditions:

Normal kidneys.
Dilatation of the pelves and ureters.
Acute degeneration of the kidney.
Acute exudative nephritis.
Acute productive nephritis.
..Chronic nephritis.

In some of the patients, even in young women, I have found well-marked disease of the cerebral arteries.

Etiology.—A number of theories have been entertained as to the causes of puerperal eclampsia. It must be confessed that no one of them is satisfactory, and that the subject is still obscure. The ordinary explanations are as follows:

1. Pressure on the renal veins by the gravid uterus produces a chronic congestion of the kidneys, which interferes with their functions.

2. Pressure of the uterus on the ureters renders it necessary for the kidneys to secrete against a higher pressure, so that they are unable to get rid of the proper quantity of excrementitious substances.

3. The kidneys are obliged to excrete waste products not only from the mother, but also from the enlarged uterus and the fœtus, and this extra work they are unable to perform.

4. It has been demonstrated in some cases that before the convulsions there is a diminished quantity of excrementitious products in the urine, and after the cessation of the convulsions an increase of these products; therefore, the convulsions are due to the retention of these excrementitious substances in the blood.

5. That for some reason the patients have cerebro-spinal congestion.

6. That for some reason they have cerebro-spinal anæmia.

7. That the convulsions are of the nature of acute epileptic attacks due to irritation of nerves in the pelvis.

8. That the enlarged uterus acts as an irritant to the vaso-motor nerves and so causes a contraction of the arteries throughout the body.

9. That the enlarged uterus causes irritation of the vaso-motor nerves which supply the renal arteries; the contraction of the renal arteries causes death or degeneration of the renal epithelium; the changes in the renal epithelium render the kidneys unable to excrete poisonous substances; the accumulation of these substances in the blood causes the convulsions, etc.

10. There is at the time of childbirth in some women a toxic substance produced in some unknown way, which is not caused by any change in the function of the kidneys, but which is capable of causing transudation of serum from the vessels, contraction of the arteries, acute degeneration of the kidneys, and acute nephritis. In other words, the changes in the kidneys are not the cause of the convulsions, etc., but they are the result of the same poison which produces albuminuria and the cerebral symptoms.

Symptoms.—1. There are a considerable number of pregnant women in whose urine during the latter months of pregnancy albumen is present in appreciable quantities; they have no other symptoms and pass through labor without trouble.

2. There are women who, during the later months of pregnancy, have scanty and albuminous urine, more or less dropsy of the legs, and become pale and anæmic. They may pass through childbirth safely and do well afteward, but some of them have a chronic nephritis dating from the pregnancy.

3. In a small number of women at about the time of childbirth, either before, during, or after labor, there are cerebral symptoms. In some of these women albumen has been present in the urine during the pregnancy; in others, besides the albuminuria, dropsy and anæmia have also been present; but in others the cerebral symptoms are suddenly developed without any premonitory conditions.

The cerebral attacks are characterized by nausea and vomiting, headache, blindness, muscular twitchings, general convulsions, stupor, coma, hemiplegia, a rise of temperature, a pulse of high tension, venous congestion of the skin, the urine diminished in quantity or suppressed, usually containing large quantities of albumen. The cases vary as to how many of these symptoms are present. A fair proportion of the patients survive these attacks, although the children usually die. In the fatal cases death takes place with the general convulsions, with hemiplegia, or with coma.

After the termination of the labor and the disappearance of the alarming symptoms the anxieties of the obstetrician are at an end, but those of the physician begin. For in many of these women a nephritis originates during pregnancy which continues afterward as a chronic inflammation, and ultimately destroys life.

Treatment.—While there have been many different opinions as to the nature of puerperal eclampsia, there is a good deal of uniformity as to the treatment. If the convulsions come on at about the end of pregnancy, it is generally agreed that labor should be brought on and the child delivered as soon as possible. Apart from this we try to unload the veins and dilate the arteries. This can be done by general blood-letting and by the use of nitroglycerin, chloral hydrate and opium. In order to guard against the cerebral symptoms it is of more practical importance to watch the arteries and the heart than to test the urine for albumen.

SUPPURATIVE NEPHRITIS.

Suppurative inflammation of the pelvis of the kidney and of the kidney itself occurs under several different conditions. It is the result of injuries; it is due to emboli; it occurs without discoverable cause; it is secondary to cystitis, the cystitis being due to stricture of the urethra, to stone in the bladder, to paraplegia, to operations on the urethra, bladder and uterus, to gonorrhœa, or to enlarged prostate. Chronic suppurative pyelo-nephritis may be caused by the presence of calculi in the pelvis of the kidney.

1. *Suppurative Nephritis from Injury.*

Gunshot wounds, incised or punctured wounds, falls, blows and kicks are the ordinary traumatic causes. If the injury be a very severe one, it causes the death of the patient in a short time; if it be less severe, suppurative inflammation may be developed.

The inflammatory process may be diffuse, so that the whole of one or both kidneys is converted into a soft mass composed of pus, blood and broken-down tissue; or it is circumscribed, and one or more abscesses are found in the kidney, which may communicate with its pelvis.

Symptoms.—Rigors mark the beginning of the suppuration and are often repeated throughout its course. A febrile movement is developed, which is apt to assume the hectic character with sweating. There is often vomiting. There may be very severe pain referred to the region of the inflamed kidney. The urine is diminished or suppressed; it contains blood alone, or blood and pus. In the bad cases the patients pass into the typhoid state, become delirious, and die comatose, or with a rapid and feeble pulse. Or the disease is protracted, the patients become more and more emaciated, and finally die exhausted. In other cases the symptoms abate, the urine returns to its natural condition, and the patients recover.

Treatment.—The management of these cases is rather surgical than medical. The external wound is to be treated antiseptically, and the suppurating kidney is to be incised or removed, as may be necessary.

2. *Abscesses Produced by Emboli.*

In ordinary endocarditis with vegetations on the valves it often happens that fragments of the vegetations become fixed in the branches of the renal artery. When this is the case white infarctions are produced.

With malignant endocarditis and with septic infections emboli find their way into the branches of the renal artery and set up circumscribed foci of suppurative inflammation. The kidneys become enlarged and are studded with little white points surrounded by red zones. These little white points are formed by an infiltration of pus cells between the tubes, followed by the death and breaking down of the kidney tissue. The bacteria of suppuration are found in these little abscesses.

Symptoms.—These embolic abscesses can hardly be said to have a clinical history. Whatever symptoms may belong to them are lost in those of the general disease from which the patient is suffering.

3. *Idiopathic Abscesses.*

These occur without discoverable cause. Only one kidney is involved. We find after death part of the kidney destroyed; the remaining portions contain abscesses; the pelvis is dilated and contains pus, the capsules are thickened, the suppurative inflammation may extend to the surrounding tissues so that sinuses are formed, and even perforations into the intestine or through the diaphragm. It is very difficult in these cases to tell whether the inflammation begins in the kidneys or in its pelvis.

Symptoms.—The symptoms begin gradually and are for some time obscure. There are repeated chills and an irregular febrile movement. The patients lose flesh and strength, become anæmic, and are often troubled by nausea and vomiting. There is more or less pain over the inflamed kidney. After a time the pelvis of the kidney may be so much dilated as to form a tumor. If the pus escapes from time to time through the ureter, this tumor will vary in size. The urine at intervals contains pus and fragments of broken-down kidney tissue. If the suppuration extends there will be sinuses running behind the peritoneum, or into the colon, or upward through the diaphragm. The disease is apt to last a long time. The patients are liable to have chronic nephritis of the other kidney, or waxy degeneration of the viscera.

Treatment.—The only plan of treatment is to cut down on the suppurating kidney and treat it as an abscess, or to remove it altogether.

4. *Suppurative Pyelo-Nephritis with Cystitis.*

One or both kidneys become inflamed. The pelves are congested and coated with pus and fibrin. The kidneys are swollen, congested and studded with foci of pus. The smallest foci are not visible to the naked eye, but with the microscope we find collections of pus cells between the tubes, with swelling and degeneration of the epithelium within the tubes. The larger purulent foci look like white streaks or wedges running parallel to the tubes and surrounded by zones of congestion. The larger abscesses replace considerable portions of the kidneys.

The ureters in some cases are inflamed, their walls thickened, their inner surfaces coated with pus or fibrin. The bladder presents the lesions of acute or of chronic cystitis.

Etiology.—This form of nephritis seems to be always secondary to a cystitis, the infection extending from the bladder through the ureters to the kidneys. The cases of cystitis in which a suppurative nephritis is likely to be developed are those due to strictures of the urethra, stone in the bladder, operations on the urethra, bladder and uterus, paraplegia, gonorrhœa, and enlarged prostate.

Symptoms.—When the nephritis occurs with cystitis due to stone in the bladder, strictures of the urethra, or operations on the genito-urinary tract, the symptoms are much the same. The patient has first the symptoms belonging to the cystitis, then he is attacked with chills and a rise of temperature. The chills are repeated, the temperature is irregular and accompanied by profuse sweating. There is a rapid change in the general condition of the patient; he becomes more prostrated and emaciated from day to day. The face is drawn and anxious, the tongue dry and brown, the pulse rapid and feeble; delirium is developed, and the patient finally dies in the septic condition. The urine is diminished in quantity or suppressed; it contains blood, pus, and mucus derived partly from the bladder, partly from the kidneys.

Cases of suppurative nephritis due to a gonorrhœal cystitis are not common, but several of them have been observed. Murchison describes two cases, in both of which the cerebral symptoms were very marked—delirium, convulsions, and coma.

When suppurative nephritis complicates the cystitis due to enlarged prostate, the symptoms are somewhat different. The patients are usually men over fifty years old. They have generally suffered from the symptoms of enlarged prostate, retention of urine either constant or intermittent, and more or less cystitis with pus and mucus in the urine. Sometimes, however, no such history is obtained; the patients assert that they have had no previous bladder trouble. The first symptom is a diminution in the quantity of urine, with the appearance of blood mixed with it, or the urine may be suppressed altogether. The blood may be present in considerable quantities, so that the patients seem to pass blood instead of urine. The patients rapidly become prostrated and very anxious. There are usually no chills, and there may be no rise in temperature. The prostration becomes more marked, the pulse is rapid and feeble, the skin is cold and bathed in perspiration, and the patients die in collapse at the end of a few days. Or, instead of such a history, the patients may behave as if they were the subjects of septic poisoning.

Prognosis.—Suppurative nephritis secondary to cystitis is a very fatal disease; so far as I know all the patients die.

Treatment.—The treatment for these cases is altogether a preventive one directed to the cystitis. When the nephritis is once established we have no further control over the case.

CASE XL. *Suppurative Nephritis, with Cystitis.*—Male, 24 years old, September 20, 1870. Three days ago he was attacked with pain in the hypogastric region. The next day he went out, the pain came on again, and he could pass no urine. That evening a catheter was passed but no urine found.

After his admission 8 ounces of urine were drawn off with the catheter.

Three days later he died.

Autopsy.—The heart and lungs are normal. The liver is large and fatty, the spleen is large.

There is a general peritonitis most marked in the lower part of the abdomen.

There is a single kindey with a single large ureter, the renal artery is given off from the aorta between the common iliac arteries. The pelvis and calyces are dilated, there are purulent foci scattered through the kidney.

The bladder is of medium size, not hypertrophied, the mucous membrane is congested with sloughy patches.

Case XLI. *Suppurative Nephritis; Stricture of the Urethra; External Urethrotomy.*—Male, 57 years old, July 22, 1876. When he was 20 years old he had a stricture which was treated and said to be cured.

For five years after this he had no trouble; after that the urine was passed slowly and in a small stream.

Five years ago he was operated on and relieved for a time.

Two and a half years ago internal urethrotomy was performed.

One year ago he had gonorrhœa, with an abscess in the perineum, which was opened.

Now he has stricture of the urethra, and an abscess in the perineum.

August 16 external perineal urethrotomy was performed.

September 5 blood was passed with the urine.

September 18 he had a chill, temperature 104°, pulse 160. After this he continued in the septic condition until he died, September 30.

Autopsy.—The brain was not examined. The lungs are emphysematous, the bronchi contain muco-pus.

The aortic valves are a little thickened.

The liver and spleen are normal. The mucous membrane of the stomach is coated with mucus.

The wall of the bladder is hypertrophied, its cavity is small, its mucous membrane is thickened and covered with muco-pus.

The ureters are dilated and contain pus; near the bladder they are blocked up with small calculi.

The pelves of the kidneys are much dilated and coated with pus. Of the left kidney not much kidney tissue is left.

There are numerous small and large abscesses in both kidneys.

Case XLII. *Suppurative Nephritis; General Sepsis.*—Male, 25 years old, October 31, 1879. Five weeks ago he had an attack of pleurisy on the right side; at the same time the urine was turbid and scalding. Since that time he has not felt well and had irregular chills.

Three weeks ago he felt so ill that he remained in bed. Three days later the urine was passed frequently in small quantities and was turbid.

Two nights ago there was retention of urine for 24 hours, followed by dribbling.

Now he is pale, emaciated, the bladder very much distended, the prostate large and tender. The urine was drawn off with the catheter; it contained pus.

November 14 an abscess formed in the right lumbar region, which was opened on November 25.

November 27 the right leg and thigh became painful and swollen, an abscess formed in the calf of the left leg.

All the time he was in the hospital he was in the septic condition, with temperature from 98° to 104°.

December 19 he died.

Autopsy.—The body is emaciated. There are old adhesions over the lower lobe of the right lung. The lower lobe of the left lung is coated with pus.

The heart and spleen are normal.

The liver is large and waxy.

The peritoneum is thinly coated with pus.

The bladder is congested and contains pus and urine, the connective tissue around it is infiltrated with pus.

The prostate is honeycombed with purulent cavities.

The inferior vena cava contains puriform thrombi.

Both kidneys are studded with large foci of pus. The capsule over the posterior surface of the right kidney is separated from it by a large abscess. The pelves of both kidneys are thickened and inflamed.

CASE XLIII. *Suppurative Nephritis; Fracture of the Spine.*—Male, 26 years old, August 7, 1885. Just before admission he was struck on the right shoulder and knocked into a sitting position. Immediately afterward there was loss of motion and sensation in both legs and the urine was retained.

Now there is complete paraplegia with retention of urine.

The urine was drawn off regularly with the catheter.

August 11 he began to have fever, and the urine was purulent.

After this he was septic, with repeated chills, a febrile movement, urine containing pus and blood, until he died, October 11.

Autopsy.—The body is emaciated. The body of the twelfth dorsal vertebra is fractured and dislocated forward.

The spinal cord is compressed and softened.

The bronchi contain pus.

The heart, liver, spleen, stomach and intestines are normal.

The wall of the bladder is thickened; its mucous membrane is congested and coated with pus and fibrin.

The ureters and pelves of the kidneys are dilated and contain pus.

Both kidneys are studded with small foci of pus.

CASE XLIV. *Suppurative Nephritis; Cystitis; Gonorrhœa.*—Male, 32 years old, December 4, 1881. He has had several attacks of gonorrhœa. Three weeks ago he began to have retention of urine and became very weak.

Now there is a purulent discharge from the urethra, the bladder is distended with urine, the man is septic.

The urine was regularly drawn off with the catheter, the man continued in the septic condition with temperatures from 97° to 104° until he died, December 25.

Autopsy.—The brain was not examined. The heart and lungs are normal. The liver is large and waxy.

The peritoneal surface of the bladder is coated with fibrin, its inner surface is studded with large, gangrenous patches.

The ureters are dilated and contained pus.

The pelves of the kidneys are congested and coated with pus. There are large and small foci of pus in both kidneys.

Case XLV. *Suppurative Nephritis, Secondary to Gonorrhœal Cystitis.*—Female, 35 years old, July 26, 1876. For two months she has had a profuse leucorrhœa with a good deal of pelvic pain.

Now she is well nourished, no appetite, tongue coated, pulse and temperature normal, urine 1.008, no albumen.

August 2 she passed into the septic condition, pulse 96 to 132, temperature 99° to 104°, and died August 7.

Autopsy.—The brain was not examined. The heart is normal. The bronchi are congested and coated with mucus, with blood clots extending into the small bronchi.

The liver, spleen and stomach are normal. On the mucous membrane of the large and small intestine there is an increase of mucus.

The uterus is large, not inflamed, with fibroids in its walls.

The bladder is small, its mucous membrane congested and coated with mucus.

The pelves of the kidneys are dilated, congested and coated with mucus. Both kidneys are studded with small foci of pus.

Tubercular Nephritis.

The different portions of the genito-urinary tract—the kidneys, ureters, bladder, seminal vesicles, prostate, testicle, uterus, Fallopian tubes, and ovaries—may become the seat of a localized tubercular inflammation.

Such an inflammation may involve one of these organs, or several of them. If several of them are involved, they are on the same side of the body, usually the left side. The inflammation is attended with the growth of tubercle bacilli and the formation of tubercle tissue. The tubercle tissue soon dies and undergoes cheesy degeneration.

In the kidneys the inflammation begins in the mucous membrane of the pelvis and calyces, and extends to the parenchyma, until a large part of the kidney is replaced by the degenerated new tissue. The cheesy masses may soften or become calcified, while the kidney tissue between them is converted into fibrous tissue more or less infiltrated with pus.

The other kidney, after a time, is apt to become the seat of the exudative form of chronic nephritis, with waxy degeneration of the blood vessels.

The tubercular nephritis may be complicated by tubercular inflammation of other parts of the genito-urinary tract on the same side of the body, by tubercular peritonitis, pulmonary tuberculosis, or general tuberculosis.

The disease is said to occur in all ages; it is most frequent in middle-aged persons. It occurs twice as often in men as in women.

Symptoms.—The urine usually, but not always, contains from time to time blood, pus, detritus, epithelium, shreds of tissue, and tubercle bacilli. When the other kidney has become the seat of chronic nephritis the specific gravity of the urine falls and albumen and casts are present.

Pain, either continuous or in paroxysms, and tenderness are often present in the inflamed kidney. There may be hectic fever with night sweats; the patients gradually lose flesh and strength. The kidney may be enlarged so as to form a tumor which can be felt. After a time there are added the symptoms of tubercular inflammation of other parts of the genito-urinary tract, of tubercular peritonitis, of pulmonary phthisis, of waxy degeneration of the viscera, or of chronic nephritis of the other kidney.

The disease lasts, as a rule, for several years. Most of the cases terminate fatally, but it is possible for the inflammation to stop and for the patient to recover.

Treatment.—The proper treatment for tubercular nephritis is the removal of the diseased kidney. The practical difficulty is to make the diagnosis before other parts of the genito-urinary tract have become tubercular, or before the remaining kidney has become the seat of chronic nephritis.

We are very much helped both in making an early diagnosis and in determining which kidney is diseased by the use of the endoscope, and by the practice of catheterizing the ureters.

We may hope that climate and feeding may have the same good effects on tubercular nephritis as they have on pulmonary tuberculosis.

Case XLVI. *Tubercular Nephritis.*—Male, 37 years old, November 24, 1882. Eighteen months ago he began to cough. Fifteen months ago he had several small hæmoptyses, two months ago a large hæmoptysis. He also at the beginning of his illness had pain over the left kidney and bloody urine. There has been bloody urine off and on ever since.

Now he is pale and emaciated, pulse 96, breathing 34, temperature 100°, urine 1.018, alkaline, contains albumen. There are dullness on percussion and subcrepitant râles over the upper lobes of both lungs.

November 26 the urine contained blood and pus. He went on like a patient with chronic miliary tuberculosis and tubercular nephritis until he died, May 12.

Autopsy.—The brain was not examined. The body is much emaciated. The lungs are covered with old adhesions. Both upper lobes are small and contracted, they are converted into fibrous tissue and miliary tubercles and contain cavities.

The liver and spleen are normal. The right kidney is normal except for a little yellow patch at the apex of one pyramid.

The left kidney is small, with very little kidney tissue left, but replaced by large, cheesy nodules. The wall of the pelvis and of the ureter is thickened and cheesy.

The bladder contains thick, grumous urine and small, soft calculi, most of the mucous membrane is destroyed, leaving a bare, ragged surface composed of poor granulation tissue in which are tubercle granula.

The ureter is completely filled with cheesy matter, its mucous membrane is destroyed, its wall is thickened.

Case XLVII. *Tubercular Nephritis.*—Male, 23 years old, October 27, 1883. He was well until two years ago. Then he was suddenly attacked with frequent and painful urination and pain in the back. For six months following, every day he passed small, soft, white masses with the urine. Then he felt well for a month. Then he felt sick again and passed the same thick, white masses with the urine.

Then there was again an improvement until two months ago, when blood appeared in the urine, and has continued ever since.

Now on the left side there is pain and tenderness in the lumbar region, extending down into the groin. Urine 1.012, contains a little blood and a good deal of pus.

November 11 he began to have fever and failed rapidly.

January 21 he died.

Autopsy.—The brain was not examined. The body is emaciated. The heart is normal.

There are large, white miliary tubercles in the upper lobes of both lungs.

The liver and spleen are normal. The stomach and duodenum are distended with brownish fluid.

The peritoneum is congested and a little roughened.

The bladder is empty, small, its wall not thickened, the mucous membrane studded with miliary tubercles.

The prostate and seminal vesicles are normal.

The right ureter and the pelvis of the kidney are a little dilated, contain pus and their walls are somewhat thickened. The right kidney is normal.

The left ureter and pelvis are much more dilated and thickened, with a growth of granulation tissue and tubercle granula. In the left kidney there are cheesy masses and miliary tubercles in the pyramids and cortex.

PYELITIS.

Inflammation of the pelvis of the kidneys occurs in a variety of ways.

1. There are the acute cases in which the pelves of both kidneys are infected from the bladder with suppurative inflammation, and the inflammation involves also the kidneys themselves. In this form the inflammation of the kidneys is more important than that of the pelves, and we class the cases with those of suppurative nephritis.

2. Calculi in the pelvis of the kidney often set up a pyelitis, which will be described with the account of renal calculi.

3. An acute catarrhal pyelitis of mild type is caused by cantharides and turpentine, and may accompany the exanthemata, typhoid fever and other infectious diseases. It usually lasts but a short time and gives no symptoms except the pus cells in the urine.

4. There is a large class of cases of pyelitis in which the inflammation is confined to one of the kidneys, is often secondary to cystitis, but yet may originate without cystitis, or other discoverable cause.

The inflammation is of catarrhal type, with the production of pus cells in variable but, in some cases, in very large quantity. The inflammation may begin in the pelvis of a kidney which is normal, of one already the seat of hydronephrosis, of one already involved in a chronic nephritis, or of one which is movable.

The mucous membrane of the pelvis and calyces is thickened, its stroma more or less infiltrated with cells, the layer of epithelium thickened in some places, thinned in others, its surface coated with mucus or muco-pus. The pelvis and calyces are more or less dilated, they may contain phosphatic concretions. In the kidney itself there may be a growth of connective tissue in the stroma of the pyramids and cortex, with degeneration of the epithelium and atrophy of the glomeruli, or a suppurative inflammation which may destroy considerable portions of the kidney.

Symptoms.—In some of the patients the symptoms of the cystitis, of the enlarged prostate, or of the stricture of the urethra are prominent features of the case, but in others there are no symptoms except those of the pyelitis.

So far as the pyelitis is concerned, the patients have pain and tenderness referred to the position of the diseased kidney. The urine from time to time contains blood, mucus, and epithelial cells from the pelvis of the kidney; later in the disease many pus-cells are mixed with the urine, and sometimes phosphatic concretions. The urine is more frequently acid than is the case with cystitis. The pus may be discharged continuously, or the ureter may from time to time become occluded, and the urine coming from the other kidney will be clear until the obstruction is overcome; then a large quantity of purulent urine will again be discharged.

The cases vary as to the severity, duration and results of the pyelitis.

1. There are cases in which the pyelitis is not very severe, the effect on the health of the patient is not considerable, the changes in the urine and the pain over the kidneys are the prominent symptoms. In these patients the inflammation may be much improved, or cured by medical treatment.

2. There are cases in which from time to time there is an increase in the quantity of pus, a rise of temperature, and evidences of septic poisoning. In some of these patients surgical interference seems unavoidable, and yet rather threatening cases seem to get well without much interference.

3. In some patients the purulent inflammation of the pelvis continues; more or less suppurative inflammation of the kidney is added; there are also progressive loss of flesh and strength, chills, hectic fever, waxy degeneration of the viscera, and the patients finally die exhausted by the disease.

4. The distention of the pelvis of the kidney may be so great as to form a fluctuating, painful tumor of considerable size. If the ureter is not entirely occluded, the pus will escape from time to time with a corresponding change in the size of the tumor.

5. Occasionally, after a time the purulent discharge ceases, the pelvis contracts, the kidney is atrophied, and the patient gets well with one useful kidney. In some of the cases the secondary nephritis after a time gives symptoms. The specific gravity of the urine falls, the patients lose flesh and strength, become anæmic, have more or less dropsy, and finally have acute cerebral symptoms or pass in the condition of chronic uræmia.

6. If phosphatic concretions are formed in the pelvis, fragments are liable to come away from time to time and cause attacks of renal colic. Or it is possible for such fragments to become impacted in the ureters and cause fatal suppression of urine.

Treatment.—The stricture of the ureters, enlarged prostate, cystitis, or other cause of the pyelitis, is to be removed as entirely as possible. For the chronic pyelitis the improvement of the general health, an out-of-door life in a suitable climate, and the use of such waters as those of Ems and Poland Springs are often of great service. A considerable number of drugs have been employed for the cure of pyelitis—the mineral acids, the tincture of the chloride of iron, the alaklies, the vegetable astringents, salol, belladonna, and others. In pyelitis in the female good results are reported from washing out the pelvis through the ureter. If the distension of the pelvis with purulent fluid is very great, or if the kidney itself undergo suppuration, the kidney must either be opened or removed. The cystoscope is of much service in locating the kidney in which the pyelitis is situated.

Hemorrhagic Pyelitis.

Cases characterized by intermittent attacks of pain over one kidney and bloody urine, or of bloody urine alone, are of not uncommon occurrence. Only a moderate number of these cases have found their way into print, but conversation with physicians and surgeons shows the existence of many unreported cases. The symptoms may follow either a mild or a severe course.

The milder form of the disease seems to be of the most common occurrence in young girls. The only marked symptom is the appearance in the urine, for days or weeks, of red and white blood cells and of epithelium from the pelvis of the kidney. The patients may also suffer from hysteria and from disturbances of digestion. After one or more of such attacks the patients recover altogether. No treatment is necessary except to relieve disturbances of digestion and improve the general health.

The severe form of the disease is seen both in males and females. From time to time the patients suffer from attacks of pain referred to one kidney, and at the same time the urine contains large quantities of blood. With the cystoscope the blood can be seen to come from the ureter belonging to the affected kidney. Some patients after a number of such attacks have no further trouble and recover entirely. In others, however, the pain is so intense, or the loss of blood so threatening, that surgical operations have been undertaken for their relief, usually with the expectation of finding calculi in the kidney. These kidneys have been simply cut down on and felt of, or needled, or split open, or extirpated. It is a curious fact that recoveries are reported after each one of these operative procedures.

In the only one of these kidneys which I have had the opportunity of examining after extirpation, the kidney tissue itself was normal and contained no blood in the tubes or stroma. The mucous membrane of the pelvis and calyces was much thickened, its stroma infiltrated with cells, and the epithelium irregular.

HYDRONEPHROSIS.

Definition.—Dilatation of the pelvis and calyces of the kidneys.

Etiology.—Dilatation of the pelvis and calyces of one or of both kidneys can be produced by any mechanical obstruction to the escape of urine.

Such an obstruction may begin during fœtal life, so that when the child is born both ureters and the pelves of both kidneys are found much dilated. Such children die soon after birth. The hydronephrosis in these cases is due to some congenital malformation, but occasionally we see this condition in children in whom it is very difficult to find the seat of obstruction. It is supposed that in these cases there exists a membranous obstruction which is broken by the probe used to explore the urethra.

In adults the mechanical cause of the obstruction to the escape of urine may be situated in the ureter, bladder, or urethra, or in the abdominal cavity near the ureters. According to the position of the obstruction, either one or both kidneys are affected.

Morbid Anatomy.—The pelves and calyces of the affected kidney are more or less dilated; the mucous membrane is thin and shining, or thickened. The kidney tissue becomes more and more thinned as the dilatation goes on, and after a time in the thinned kidney a chronic nephritis is set up. Then there is a growth of new connective tissue in the stroma of the pyramids and cortex:

in some of the glomeruli there is a growth of the cells covering the capillaries, while other glomeruli are atrophied; degeneration of the renal epithelium and thickening of the walls of the arteries are also present.

Symptoms.—The patients suffer for a considerable length of time from the inconveniences belonging to the retention of urine. If the retention be due to stricture of the urethra or to disease of the bladder, the entire history is straightforward. Obstructions of the ureters, however, may give no evidence of their existence. and we find after death strictures of one or both ureters for which we are unable to account.

It is not uncommon to find after death well-marked hydronephrosis of one of both kidneys in patients who have had no renal symptoms at all.

In some cases, after a time, there is developed in the compressed kidneys a chronic productive nephritis with a little exudation. Then the specific gravity of the urine gradually falls, a small quantity of albumen and a few casts make their appearance. The quantity of the urine remains normal, or is increased, or diminished, or suppressed. The patients may at any time have contraction of the arteries with an elevated temperature and cerebral symptoms, or they may pass into the condition of chronic uræmia with a feeble pulse and low temperature.

In other cases the most prominent symptom is the presence of a tumor in the abdominal cavity. The disease is then usually confined to one kidney. The tumor lies in the region of the loin, extending upward, downward and forward as it increases in size, and pushing the intestines forward and to one side. The surface of these tumors may become inflamed, and adhesions formed to the surrounding tissues. As they become larger they give rise to much discomfort and pain. The tumors feel like cysts. From time to time they may diminish in size, with an increased flow of urine from the bladder; they are not movable. If the disease is caused by renal calculi, there may be attacks of renal colic with bloody urine. If the fluid from the tumor is drawn off it usually contains urinary salts, but sometimes it is only clear serum.

In still other cases of hydronephrosis a suppurative inflammation attacks the pelvis of the kidney, and the patient suffers from pyelitis.

Treatment.—The treatment of hydronephrosis is altogether surgical. If possible the cause of the retention of urine must be removed. If large cysts are formed, they must be opened or removed.

RENAL CALCULI.

The solid constituents of the urine may be precipitated in the pelves of the kidneys in the form of sand, gravel, or calculi. The calculi are formed of uric acid, of uric acid with a shell of oxalate of lime, of oxalate of lime, or of the phosphates. Calculi composed of cystin are of a light-yellow color and lustrous, looking something like beeswax; they are of rare occurrence. Calculi composed of xanthin, of fatty or saponaceous matters, of carbonate of lime and of fibrin have been described in rare cases.

Etiology.—Renal calculi are found in persons of all ages; they are of more frequent occurrence in males than in females. A sedentary life; gout; any conditions which produce an exaggerated excretion of uric acid, of oxalate of lime, of phosphates, or of cystin, or inflammation of the pelves of the kidneys, may act as causes.

Symptoms.—Small calculi may be formed in the pelves of the kidneys, be passed through the ureters into the bladder, and finally escape through the urethra without giving any trouble.

Larger calculi, which are formed in the pelves and pass through the ureters, cause by their passage attacks of renal colic. During an attack of renal colic the patients have severe pain, coming on suddenly or gradually, referred to the position of the kidney and ureter and radiating down into the groin, or simply a diffuse abdominal pain. The testicle is retracted, sometimes painful and swollen. Occasionally the pain is so severe that the patients faint or have general convulsions. Vomiting and retching may be troublesome and distressing symptoms. There may be a moderate rise of temperature.

The urine, during the attack, is passed frequently, in small quantities, often with a good deal of pain, and may contain blood. After the attack the urine is passed in considerable quantities, and may contain blood.

Most attacks of renal colic only last for a few hours, but they may be protracted, with intermissions, over a number of days. The same patient may have one attack, or several attacks.

The calculus, instead of passing through the ureter into the bladder, may become impacted. If the patient has only one kidney, or if the ureters of both kidneys are obstructed, the urine is entirely suppressed and the patients die after a number of days. It is noticeable that such a calculus suppression of urine causes death without any active uræmic symptoms. If the ureter of only one kidney is obstructed, either hydronephrosis or pyonephrosis is apt to follow.

The calculi, after being formed in the pelvis of the kidney, may remain there. When this happens the patient may never have any symptoms, nor any considerable change in the kidney; but without any symptoms the kidney may become atrophied. More frequently the patients have repeated attacks of pain over one kidney and bloody urine. After a number of attacks they may finally pass a calculus. If the attacks cease and no calculus is passed we cannot be certain whether the patient has a renal calculus, or has had a hemorrhagic pyelitis.

Very often the calculus acts as an irritant and sets up inflammatory changes in the pelvis or in the kidney. Or instead of this, a pyelitis is followed by the formation of phosphatic calculi.

If there is a chronic pyelo-nephritis, the patients have pain and tenderness over the diseased kidney either continuously, or in attacks. The urine at intervals contains pus and blood, sometimes fragments of the calculi. The dilatation of the pelvis of the kidney may form a tumor of appreciable size. The patients lose flesh and strength, they have an irregular fever, they are liable to have chronic nephritis of the other kidney, or waxy degeneration of the viscera.

In some cases the calculus causes but little change in the pelvis of the kidney, but the kidney itself becomes the seat of chronic diffuse nephritis with a little exudation. The urine then becomes of low specific gravity and contains a little albumen. The patients are liable to attacks of contraction of the arteries, with dyspnœa or cerebral symptoms, or they may become somewhat dropsical.

Treatment.—We insist on sufficient exercise in the open air. The patients should spend several weeks every year at one of the alkaline springs, or should take from time to time one of the alkalies largely diluted with water. Piperazin is recommended in doses of from five to ten grains dissolved in large quantities of water. Urotropin is also given in doses of twenty grains daily, dissolved in water and taken at one dose in the morning.

The treatment of the attacks of renal colic is directed to the relief of the pain and to hastening the passage of the calculus. The means usually employed for this purpose are hypodermic injections of morphine, inhalations of ether and chloroform, and the hot bath. The use of doses of thirty drops of tr. belladonnæ every two or three hours, until slight delirium is produced, has been employed to cut short the attacks and favor the passage of the calculus.

A calculus in the pelvis of the kidney, or one impacted in the ureter, can only be removed by a surgical operation.

THE CYSTIC KIDNEY.

Cysts are formed in the kidney both during intra-uterine and extra-uterine life.

The congenital cystic kidney is a very remarkable pathological condition. Either one or both kidneys are enormously enlarged and converted into a mass of cysts. The cysts are of all sizes, and are separated from each other by fibrous septa or compressed kidney tissue. They contain a clear, yellow, acid fluid, holding in solution the urinary salts; or the fluid is turbid and brown, and contains blood, uric acid crystals and cholesterine. The cysts are lined with a single layer of flat epithelial cells. They seem to be formed by a dilatation of the tubules and of the capsules of the Malpighian bodies. As causes for such a dilatation are found obliteration of the tubes in the papillæ; or stenosis of the pelvis, ureters, bladder or urethra. Other congenital malformations are often associated with this one.

In adult life we find three varieties of cystic kidney:

1. In kidneys, which are otherwise normal, there are one or more cysts filled with clear or brown serum, or colloid matter. These cysts do not appear to interfere at all with the functions of the kidneys.

2. In chronic diffuse nephritis, especially in the atrophic form, groups of tubes are dilated. Apparently one or more of the larger tubes in the pyramids are obstructed, and this causes dilatation of a corresponding group of tubes. Such a dilatation may be moderate in size, or it may form cysts visible to the naked eye.

3. Both kidneys are very much enlarged, and converted into a mass of cysts containing clear or colored serum, or colloid matter. The nature of these cysts is uncertain. It is possible that they are congenital. They are sometimes associated with similar cysts in the liver. Their clinical history resembles that of some of the cases of chronic diffuse nephritis without exudation. The patients go on for a long time without symptoms, except that the specific gravity of the urine gradually falls and a little albumen is occasionally present. Finally they have an attack of general convulsions or of coma, and die in a short time.

NEW GROWTHS OF THE KIDNEY.

The most important new growths of the kidney are those which belong to the classes of sarcoma and adenoma.

The sarcomata grow from the kidney itself, or from its pelvis. They are composed of connective tissue with an excess of cells, with which may be mixed mucous tissue or muscular tissue. These tumors often reach a large size and may grow for a number of years before they cause death. They form a hard abdominal tumor which at first retains the position and shape of the kidney, but may finally become so large as to occupy a considerable part of the abdominal cavity.

They are found as congenital tumors, are rather frequent in infants and children, and are occasionally met with in adults.

The adenomata grow in the cortex of the kidney in the form of nodular tumors. They may follow the papillary or the tubular type. In some cases the tumor or tumors never attain any considerable size, are not malignant, and give rise to no symptoms. In other cases the tumors become much larger, and may then behave like malignant growths. These large tumors are very vascular. The adenomata which run a malignant course, with the formation of metastatic tumors, are often called carcinomata.

Symptoms.—The sarcomata and adenomata, so far as their symptoms are concerned, may conveniently be described together. In both of them there are four principal symptoms: a tumor, pain, hæmaturia, and loss of nutrition.

The tumor is appreciable as soon as it has reached a sufficient size. While it retains the natural position and outlines of the kidney the diagnosis is comparatively easy, but as the tumor becomes larger and adhesions are formed, it becomes more difficult to distinguish it from other abdominal-tumors.

Hæmaturia is present at some time in the disease in about half the cases. The hemorrhages may be very large, with rapid anæmia and exhaustion, or moderate in quantity, or so little as only to be appreciable with the microscope. They are apt to recur at intervals of days or weeks.

The pain is referred to the situation of the diseased kidney. It is by no means a constant symptom, but it may occur early and be throughout a prominent feature. It is apt to come on in attacks and to radiate downward along the course of the ureter.

Loss of appetite, nausea and vomiting are troublesome symptoms in some of the patients.

The loss of flesh surely comes sooner or later, but it is curious in some cases how long the general health may remain unaffected and how long life can be prolonged even with enormous tumors.

Treatment.—The same rule seems to hold good for these tumors in the kidney as for the same tumors in other parts of the body. If the kidney be removed while the growth is still small, the prognosis is fairly good. If it be not removed until the tumor is large or until metastatic tumors have been formed, the prognosis is bad.

LECTURES

ON

THE PRACTICE OF MEDICINE

WITH CASES AND CHARTS

BY

FRANCIS DELAFIELD, M.D., LL.D. (Yale)

PART V

TYPHOID FEVER

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TYPHOID FEVER.

An infectious disease characterized by the presence and growth of a specific bacillus, which circulates in the blood and is widely distributed throughout the body. The bacillus produces a toxin of considerable virulence and also acts as a local cause of inflammation and necrosis.

Lesions.—The typhoid bacilli seem to be regularly introduced into the ailmentary tract through the mouth and then find their way to the blood and tissues through the wall of the small intestine. As to what the bacilli are doing and where they are living during the two weeks between the time of their introduction into the body and the beginning of symptoms we have no exact knowledge.

The bacilli are regularly found in the blood early in the disease and continue till the end of the third week. Coleman and Buxton found them in the first week in 93 per cent. of 85 cases; in the second week in 76 per cent. of 198 cases; the third week in 56 per cent. of 115 cases; in the fourth week in 32 per cent. of 55 cases.

The lymphatic glands in the wall of the small intestine and those of the mesentery are the portions of the body which most frequently show inflammatory and necrotic changes.

By the second day of the disease the solitary and agminated glands of the lower part of the small intestine are swollen and the swelling increases during the first week. In the second week there is more or less necrosis of the swollen glands with sloughing and ulceration. In the fourth week there is cicatrization of the ulcers.

The minute changes which take place in the development of the intestinal lesion are as follows:

At first the blood vessels around the lymph nodules are swollen and the epithelium may fall off. Then the nodules increase in size, largely from a growth of new cells.

This cell growth is a hyperplasia of the lymph cells and the endothelium of the trabeculæ and sinuses. The production of new cells is not confined to the nodules, but extends also to the adjacent mucous membrane. There may also be little foci of similar cells in the muscular, subserous and serous coats.

In this stage resolution may take place, then the new formed cells degenerate and disappear. In the more severe cases necrotic changes supervene, by which small or large ulcers are formed.

A number of the glands are attacked at the some time, or first those in the lower part of the ileum, and later those higher up.

The lesions may be confined to the agminated glands, or involve also the solitary glands, or extend to the solitary glands of the colon.

The cases vary as to the number of glands involved, the degree of the swelling, the extent of the necrosis, the size and depth of the ulcers. In rare cases there are no changes in the small intestine.

In New York, in the fatal cases I have found the regular lesions of the solitary and agminated glands of the ileum were in most cases fairly developed. Some of the patients died while these glands were only swollen, some while the sloughs were adherent, some after the ulcers were completely cleared off. But in a considerable number of the fatal cases the intestinal lesion was inconsiderable, only a few swollen agminated glands with partial ulceration.

The glands of the vermiform appendix may be inflamed in the same way and in one case I have seen perforation.

Swelling of the mesenteric glands seems to be constant, the swelling corresponding to the degree of the intestinal lesion. When the glands are much swollen there may be a little fibrin on the peritoneum over them; rarely this extends to be a general peritonitis. In a few cases the glands suppurate, soften, and there is localized or general peritonitis.

In the colon there may be swelling and necrosis of the lymph nodes, a general catarrhal colitis, or a croupous inflammation of the lower end of the colon.

As regards the position of perforations, Fitz gives a collection of 167 cases. The perforation was in the ileum in 136 of the cases, in the colon in 20 cases, in the appendix in 5 cases, in the jejunum in 2 cases.

Peritonitis is most frequently due to perforation; it may be caused by inflammation of the mesenteric glands or occur without discoverable cause.

The spleen is always soft; it is usually enlarged, but it is important to remember that it may remain of normal size, or even be small. I have seen one case of rupture of the spleen with large extravasations of blood in the peritoneal cavity.

In the dura mater there may be thrombosis of the venous sinuses. It is said that an acute inflammation of the pia mater with the presence of typhoid bacilli sometimes occurs.

A catarrhal inflammation of the pharynx is nearly constant.

Ulceration of the larynx is described by the older authors, but is now seldom seen.

In the same way suppurative inflammation of the parotids, which used to be rather frequent, is now seldom seen.

A moderate degree of catarrhal bronchitis is present in a large number of the cases. Less frequently we find most of the smaller bronchi largely filled with pus.

Congestion and œdema of the dependent portions of the lungs, broncho-pneumonia and lobar pneumonia are of not infrequent occurrence. Gangrene and abscesses of the lung are occasionally seen.

The heart may remain normal or the muscular fibres may undergo degeneration. An acute endocarditis with the presence of typhoid bacilli has been described. I have seen one case in which I believed the patient died from a thrombus in the left ventricle, but without autopsy.

Inflammation of the arteries with the formation of thrombi has been described; it may cause gangrene.

Thrombosis of the veins occurs quite frequently; the femoral vein is the one most frequently occluded.

The most important change in the liver is the inflammation of the gall bladder. Typhoid bacilli are found in the bile within the gall bladder in the majority of the cases of typhoid fever.

The suppurative inflammation of the gall bladder belongs to the later weeks of the fever and the period of convalescence. Whether the inflammation is caused by the typhoid bacillus alone, or whether some other irritant must be added, is uncertain.

The ordinary lesion of the kidney is an acute degeneration which does not seriously affect the renal epithelium. In cases, however, which go on for 10 weeks or longer, the destruction of the epithelium is considerable. Much less frequently the kidneys show the changes of acute exudative nephritis.

Inflammation of the bladder with a good deal of pus in the urine, but without much change in the wall of the bladder, is seen quite often. Typhoid bacilli are found in the urine throughout the disease; for the production of the cystitis the addition of some other irritant seems to be necessary.

Purulent inflammations of the periosteum belong to the later stages of the disease.

There is degeneration and sometimes rupture of the voluntary muscles, especially those of the anterior abdominal wall.

Etiology.—Typhoid fever prevails in the form of local epidemics, as an endemic disease, and in the form of isolated cases. It is one of the principal causes of deaths in armies.

The history of a local epidemic is apt to be as follows: A person already suffering from typhoid fever goes to a house in the country, or in a village where the disease has not previously existed. He goes through the disease at this place. His fæces are put in a privy vault, or cesspool; his soiled clothes and bedding are washed without being disinfected. The persons who handle and wash the soiled clothes often contract the disease. The contents of the infected privy or cesspool after a time drain into the adjoining well; most of the persons who drink this contaminated water have typhoid fever. The persons living in the same village who do not drink the contaminated water, nor handle the infected clothes, do not have the disease.

Occasionally the disease is given by milk which has been contaminated by the infected water.

In some places typhoid fever is an endemic disease, prevailing year after year. When this is the case the disease is regularly much more prevalent in the autumn months, and varies in different years as to the number of cases and the type of the disease.

The most puzzling cases to account for are the isolated cases which occur from time to time in cities.

The most straightforward way for conveying typhoid bacilli into the human body is by drinking contaminated water, or milk. Less frequently they are introduced in the food. We are obliged to admit that the disease is also communicated from person to person by methods of which we are ignorant, and that it occurs in isolated cases from causes which we are unable to discover.

Symptoms.—Agglutination test, Widal's test. This is ordinarily done by drying a drop of blood from the patient on a slide, dissolving the dried blood in a little water and then mixing it on the slide with a broth culture of the typhoid bacillus; within fifteen minutes the bacilli should clump together and become motionless. Different observers get positive reactions in from 70 to 90 per cent. between the fourth and seventh day; during the second week it is more constant; it may persist for several months after convalescence. It must be admitted that there are typical cases of typhoid fever which do not give a positive result with the Widal test at any time.

The ordinary period of incubation is two weeks, with variations of a few days on either side of this limit. During this period the patient may feel well, or be troubled with headaches, disturbances of digestion, feelings of lassitude, diarrhœa, chilliness, or bronchitis.

The method of invasion varies considerably in different cases.

In a considerable number of cases one or two weeks elapse before the patients are sick enough to go to bed. They are more or less miserable with headache, sleeplessness, nausea, an irregular febrile movement and sometimes diarrhœa. In these cases it is difficult to determine the first day of the disease; we usually reckon it from the day the patient goes to bed.

In New York the ordinary invasion is rapid, although the rapid invasion is often preceded by a period of one or two weeks during which the patient does not feel well; the patients are sick enough to go to bed within twenty-four hours. The first symptoms are: one or more chills followed by a rapid rise of temperature, often accompanied with headache, vomiting, nose bleed, abdominal pain, and occasionally with diarrhœa. In a moderate number of cases the first syptoms are diarrhœa and fever.

Occasionally it happens that while the patient becomes quite ill, with a rise of temperature, during the first twenty-four hours, yet for the ten succeeding days there are intermissions of low temperature and subsidence of symptoms. After this the patient settles down into the regular course of the disease.

It may also happen that the patient begins with the regular invasion of a mild typhoid and goes on to be moderately sick for two weeks. At the end of that time the symptoms change for the worse and the disease runs a severe course.

The invasion of the symptoms may be unusually rapid and severe. The temperature runs up to 104° F., with chills within the first twenty-four hours. Here the infection is evidently very rapid and extensive.

There are irregular invasions for which we are unable to account, and which simulate the invasions of other diseases.

In some patients the first symptoms resemble those of cerebro-spinal meningitis: headache, pain, and stiffness in the back of the neck, violent delirium or general convulsions.

In some patients the complicating inflammations of the chest are very marked. It may be a bronchitis, or a broncho-pneumonia, or a pleurisy.

Or, instead of this, there is an acute catarrhal gastritis with vomiting and diarrhœa; or an acute nephritis with the characteristic changes in the urine.

During the first week there are the characteristic temperature, epistaxis, pharyngitis, headache, vertigo, apathy, sleeplessness, pain in the neck, back and abdomen, the coated tongue, loss of appetite, nausea, vomiting, diarrhœa or constipation, blood with the stools, prostration.

During the second week of the disease there are the characteristic temperature, the increased rapidity of the heart's action, more apathy, delirium, noises in the ears, deafness, the dry tongue, the eruption, diarrhœa or constipation, tympanites.

At the end of the second week there may be a temporary fall of temperature, the disease may terminate in recovery, or the patient may die.

During the third week there are present: the characteristic temperature, rapid heart action, more marked cerebral symptoms, the full development of the typhoid state, bleeding from the intestinal ulcers, peritonitis, involuntary passage of fæces, retention of urine. At the end of the third week all the symptoms may continue unabated, there may be a subsidence of the symptoms for one or more days and then the disease goes on again, or convalescence may begin.

In the fourth week, if the case is a favorable one, there is a gradual abatement of all the symptoms and convalescence commences in the fifth week. If the disease is to be protracted the symptoms of the fourth week will continue like those of the third, or there will be an improvement for one or two days and then the disease goes on again.

CASE I. (Figure 1.) *Gradual Invasion; Treatment by Intestinal Antiseptics; Four Weeks' Typhoid; Scraped Beef on the Twenty-ninth Day; Recovery.*

Female, 30 years old, trained nurse; taken sick while in the hospital. For about a week she had malaise, headache, chilliness, no appetite, abdominal pain, constipation and tympanites.

December 29 she went to bed, and this seemed to be the first day of the disease. The temperature chart begins on December 30, the second day of the disease.

Third day: Face flushed, tongue coated, abdomen tympanitic, urine 1014, no albumin, subcrepitant râles over both lungs. She was put on the use of guiacol and castor oil at first every hour, then every two hours.

Sixth day: The castor oil and guiacol have been continued until to-day. Now she is given 1-20 grain of calomel every fifteen minutes.

Ninth day: The calomel has been continued, except when she was sleeping, until to-day; now she is given Woodbridge, two and three every two hours.

Twenty-first day: She is doing very well; medicines are stopped.

On the twenty-ninth day she began with scraped beef.

On the thirty-fifth day she was out of bed.

On the forty-sixth day she was discharged.

CASE II. (Figure 2.) *Acute Invasion; Two Weeks' Typhoid; No Treatment; Recovery.*

Male, 25 years old; two days before the invasion of the disease he had mild chills, headache, body pains and malaise. The next day he felt better and went down town.

First day: He had a sharp chill in the morning, followed by sweating and prostration; a few sonorous râles over both lungs.

Second day: Vomited; the spleen could be felt.

Third to tenth day: A troublesome cough with the physical signs of bronchitis; hæmoglobin 90 per cent.; red cells 4,960,000, white cells 4,800; urine 1028, a trace of albumin, hyaline and granular casts. He had no medicine, no baths, no whiskey.

Thirteenth day: Widal reaction positive.

Eighteenth day: Had scraped beef.

Twenty-first day: out of bed.

Twenty-fourth day: Went home.

Fortieth day: Hæmoglobin 95 per cent.

Fifty-seventh day: Hæmoglobin 58 per cent.; put on sulphate of iron.

Seventieth day: Hæmoglobin 90 per cent.

CASE III. (Figure 3.) *Acute Invasion; Four Weeks' Typhoid; Diarrhœa; Treatment with Warm Baths; Recovery.*

Male, 28 years old. Had been in his ordinary health except for a little malaise for several days.

First day: He did not go down town. I saw him that evening and thought he was beginning a typhoid fever, but the temperature was not taken.

Second day: The temperature chart begins. He was given baths at 99° F. at 12 M., 4 P.M. and 8 P.M. These baths were continued regularly until the fifteenth day.

Third day: The diarrhœa began, and continued until the sixteenth day; three to seven stools daily. He was given for the diarrhœa at first cerium oxalate and bismuth, later morphine, cubebs and bismuth.

Twenty-first day: Having previously taken only milk, he was given scraped beef.

Twenty-sixth day: He was given coffee, eggs and bread for breakfast, beefsteak and potatoes for dinner.

Thirty-third day: He was out of bed.

Throughout his illness he was cheerful and comfortable and slept well every night. The convalescence was rapid.

CASE IV. (Figure 4.) *Acute Invasion; Four Weeks' Typhoid; Cystitis; Treatment with Warm Baths; Recovery.*

Female, 40 years old; for a month has been nauseated in the morning and has vomited occasionally. For a week has been more miserable, chilly, pharyngitis, pains in back and limbs.

The chart begins on the day before the invasion, when she was miserable, but had not gone to bed.

First day: Chill, vomiting, prostration; went to bed.

Second day: Highest temperature of the disease; headache, apathy, nausea, tympanites.

Fourth day: Began with tubs at 99° at 12 M., 4 P.M., and 8 P.M.; these were continued up to the sixteenth day.

Fifth day: It was necessary to draw off the urine with the catheter.

Sixth day: Positive Widal reaction.

Nineteenth day: Began to have cystitis.

Twenty-second day: Scraped beef.

Twenty-fourth day: Cystitis is well.

Twenty-eighth day: Two solid meals; out of bed.

The convalescence was uninterrupted.

CASE V. (Figure 5.) *Invasion with Repeated Chills; Low and Irregular Temperature; Rapid Pulse; Death on the Nineteenth Day; Small Intestinal Lesion.*

Female, middle aged. For five days she has had chills, fever, and sweating every afternoon and the bowels have been loose.

She came into the hospital with a tongue highly coated, no appetite, diarrhœa, urine 1021, no albumin, no physical signs over the heart or lungs.

Twelfth day: She was fairly in the typhoid state, with dry tongue, stupor, and delirium, but with low temperature.

Nineteenth day: She died rather suddenly.

Autopsy.—The brain was not examined. The heart was small but not soft. The lungs, liver, and kidneys were normal. The spleen was moderately large and soft. The mesenteric glands were a little swollen.

At the lower end of the ileum a few agminated glands were somewhat broader and thicker than normal.

CASE VI. (Figure 6.) *Invasion with Bronchitis; Three Weeks' Typhoid; Irregular Temperature; Recovery.*

Male, 30 years old. For one month dyspnœa, cough with mucous sputum streaked with blood, weakness and general malaise.

Two days ago nose bleed, chill, fever, and sweating.

The temperature begins on the third day of the typhoid fever. At that time the breathing was quick, shallow, and difficult; there were coarse and subcrepitant râles over both lungs; the tongue was brown and dry; the bowels were constipated.

The only treatment was carbonate of ammonia and whiskey.

He began to improve on the seventeenth day.

CASE VII. (Figure 7.) *Invasion with Pleurisy with Effusion; Four Weeks' Typhoid; the Patient was kept sick with Whiskey and bad Treatment.*

Female, 30 years old; hospital nurse. A week ago she began to have a dry cough, with loss of appetite, headache, chilliness, and general pains. Yesterday she began to have severe pain in the right side.

First day: Went to bed; tongue coated and moist; subcrepitant friction sound over the lower lobe of the right lung.

Third day: There were the physical signs of fluid in the lower part of the right chest.

Seventh day: The fluid had gone.

Thirteenth day: Was put on half an ounce of whiskey every three hours.

Fourteenth day: Complained of headache; was given one drachm of whiskey every hour.

Seventeenth day: Became delirious.

Twentieth day: Delirium continues; two drachms of whiskey every three hours.

Twenty-first day: Delirium and muscular twitchings; three drachms of whiskey every three hours.

Twenty-second day: Pulse said to be failing: half an ounce of whiskey every three hours; caffein, hypodermics of digitalis.

Twenty-fifth day: Vomiting, involuntary fæces and urine; whiskey continued, sodium phosphate, codeine.

Twenty-sixth day: She was in a deep stupor; whiskey continued; 100 c.c. of clear serum aspirated from right chest.

Twenty-eighth day: Continues with stupor, delirium and whiskey.

Thirtieth day: Hypodermics of digitalis. She continued in the same condition of alcoholism until the thirty-ninth day, when the whiskey was stopped.

After this she rapidly improved, and was out of bed on the forty-seventh day.

This woman began to have alcohol poisoning on the seventeenth day of her disease, and continued to have it up to the thirty-ninth day. The typhoid fever ran its course by the twenty-eighth day.

CASE VIII. (Figure 8.) *Invasion with Vomiting and Diarrhœa.*

Female, 19 years old, after two weeks of general malaise, began to have repeated vomiting and diarrhœa. At the same time prostration, headache, body pains, and fever were present. The diarrhœa was better after four days, but the vomiting continued steadily for six days. After that the symptoms were those of an ordinary typhoid terminating in recovery. The temperature chart begins on the seventh day. She was treated with cold tubs from the seventh to the twenty-fourth day.

CASE IX. (Figure 9.) *Invasion with Acute Exudative Nephritis; Treatment by Woodbridge Method; Three Weeks Typhoid; Recovery from Nephritis and Typhoid.*

Male, 24 years old.

First day: Chill, headache, bloody urine.

Second day, urine 1020, large quantity of albumin, blood and granular casts. He looks moderately sick, but not so much like typhoid fever.

Sixth day: The man now looks like an ordinary typhoid; he is put on the Woodbridge plan of treatment.

Tenth day: Urine 1014, not much albumin, very little blood.

After this the urine became normal.

Seventeenth day: His mouth was a little sore.

Twenty-first day: He was given a chop, and the medicines were stopped.

CASE X. (Figure 10.) *Invasion Like that of Cerebro-Spinal Meningitis; Death on the Seventeenth Day; Deep Ulcers in the Ileum; Peritonitis without Perforation.*

Male, 15 years old. He was said to have been in his usual health until eleven days before his admission. Then he suddenly dropped in the street unconscious, and has been in a condition of alternating delirium and stupor ever since. On the first day there was nausea and vomiting. He also had headache, fever, and marked prostration.

On admission the pupils were dilated, there was photophobia, the tongue was coated and dry, the pulse rapid and weak, there was rigidity and tenderness of the back of the neck, strabismus, general hyperæsthesia, alternating stupor and noisy delirium; the urine contained albumin and granular casts. The whole picture was that of cerebro-spinal meningitis.

Thirteenth day: Although the delirium and stupor continued, the abdomen became distended, and he looked more like typhoid fever. He was ordered tub baths at 65° F. every three hours if the temperature was over 103°. These baths continued up to the sixteenth day; the chart shows their effect on the temperature; they were very badly borne.

He became steadily worse, the stupor and delirium continuing, and died on the seventeenth day.

Autopsy.—The heart was normal, both endocardium and muscle.

In both lungs there were small patches of broncho-pneumonia.

The liver was somewhat swollen.

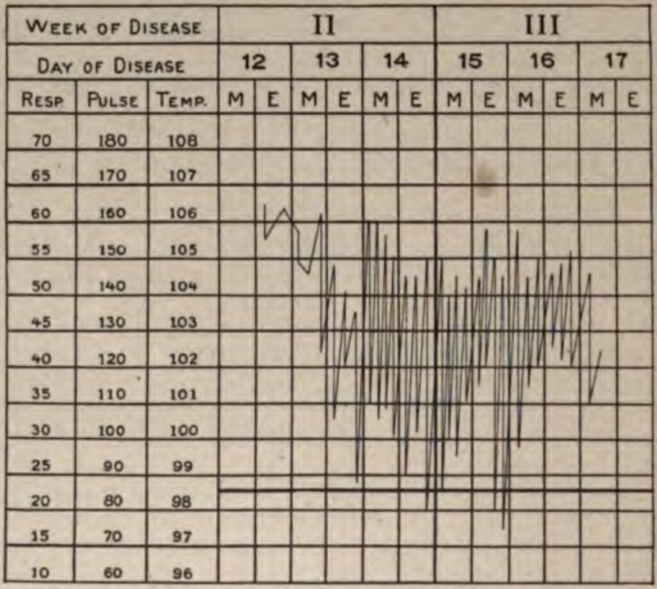
The spleen was of normal size and firm. The kidneys were of ordinary size, the cortex whitish.

The mesenteric lymph nodes were swollen.

In the ileum there were extensive and deep ulcers of the agminated glands.

In the cæcum there were two ulcers. The peritoneum over the ileum and spleen was coated with fibrin.

FIG.10



Case XI. *Two Weeks' Typhoid Fever in a Child; Cerebral Symptoms.*

Male, 5 years old. Eight days before admission pain in the neck and left ear; would not eat or play; had fever and a little cough; no diarrhœa; became more and more stupid as the days went on. Now, his face is flushed, skin hot, temperature 102°, pulse 160, breathing 34, lips and tongue dry and coated, diarrhœa, is quiet and stupid. His temperature continued between 100° and 104° until the seventeenth day, when it fell below the normal and convalescence was established. The pulse was rapid, 120 to 160. He lay quietly in bed all the time, as if asleep, like a child with tubercular meningitis. The bowels were moderately loose. The whole picture was that of a child in the typhoid state with the stupor of meningitis. Convalescence was rapid and uninterrupted.

Case XII. (Figure 11.) *Mild Typhoid Fever for Two Weeks; Temporary Improvement, then More Severe Typhoid for Three Weeks; Recovery.*

Male, 19 years old; hospital orderly. For four days headache, sleeplessness, loss of appetite, pain in abdomen, constipation, but continued at work.

First day: Moderately sick with fever, coated tongue, and prostration; was ordered tub baths at 70° F. every three hours, if the temperature reaches 103° F. After the fourth day the temperature was below the bathing point and the tubs were stopped.

Thirteenth day: Apparently convalescent.

Nineteenth day: The temperature is rising and the patient is again ill.

Twentieth day: The cold tubs are resumed and continued until the twenty-third day.

From the twentieth to the twenty-seventh day the patient was much sicker than during the first two weeks.

By the thirty-fifth day the fever was over, and on the forty-first day he was out of bed.

Case XIII. (Figure 12.) *Irregular Invasion; Irregular Course; Purulent Bronchitis; Death.*

Female, 15 years old, August 28, 1875. One week before admission she says that a man took improper liberties with her person. Several days later she had severe pain in the vulva. She now has two chancroids on the inner side of the labia majora.

September 3: She is feeling better; out of bed and walks about the ward.

September 15: She has been about the ward since September 3, but has been dull, listless, and feeble. Yesterday she said she had a diarrhœa. In the afternoon she had a chill, pain in the side, prostration, sibilant and sonorous râles over both lungs and was put to bed.

September 16, which was apparently the second day of her typhoid fever, the chart begins. It will be seen that the temperature reached 106° F., the pulse 120, the breathing 36. There are coarse and subcrepitant râles all over both lungs.

September 17, third day: There is diarrhœa; cough with mucous sputum.

September 18, fourth day: The râles are finer; the bronchitis seems to be extending into the smaller tubes; she is very weak.

September 22, eighth day: The bronchitis is still very marked; there is a typhoid eruption on the abdomen; she is very hungry.

She continued to get worse, was delirious, and died on the seventeenth day.

Autopsy.—The brain was normal.

The heart was normal.

The large and small bronchi down to the smallest were filled with pus.

The liver and kidneys were normal.

In the ileum there were deep ulcers of the solitary and agminated glands.

THE COURSE OF THE TEMPERATURE.

The chart of the temperature of typhoid fever given by Wunderlich, and followed ever since by most authors, has been the cause of numberless errors in diagnosis. It is stated that the regular course of the temperature is as follows:

During the first week the temperature rises regularly from day to day, always with a lower temperature in the morning and a higher temperature in the evening, but with each day the difference between the morning and evening temperature becomes less.

During the second week the temperature is always high, with but little difference between the morning and evening temperature.

In the third week the morning temperature begins to fall, the evening temperature remaining the same.

In the fourth week the morning temperature falls still lower, then the evening temperature falls, until finally both get down to the normal.

The maximum temperature is reached between 5 P.M. and 12 M. The minimum temperature is attained between 6 and 8 A.M.

This description does not apply to typhoid fever in the United States. It is because physicians expect such a course of the temperature that so many patients are treated for malaria during the first weeks of a typhoid.

The regular fever of typhoid is a fever that lasts four weeks, with periods of improvement at the end of the second and third weeks.

During the first week the temperature runs up slowly, or rapidly, with or without chills, the afternoon temperature higher than that of the morning.

During the second and third weeks the temperature remains high, without great variations between the morning and evening.

During the fourth week there is a gradual fall, first of the morning, later of the evening, temperature.

Exceptions to this regular course of the fever are of ordinary occurrence.

Chills, followed by fever and sweating, may be repeated for several days.

Throughout the disease there may be an unusual difference between the morning and evening temperature. The morning temperature may fall to or below the normal. In patients who have this disposition to low morning temperatures, cold tubs and antipyretics have an unusually decided effect.

In the second and third weeks there may be very little difference between the morning and evening temperatures. Rarely the morning temperature is higher than the evening. Throughout the second and third weeks there are irregular rises and falls of temperature for which we are unable to account. At the end of the second week there is apt to be some fall of temperature. This may not amount to much, or it may be very decided; so much so that there is no fever for several days. Then the fever goes up again, and may even be higher than it was during the first two weeks. In a fair number of cases the typhoid fever only lasts two weeks, and the patients become convalescent.

In the same way, at the end of the third week, there is regularly a fall of temperature, which may be exaggerated. When in this way the fever falls for several days and then goes up again the patients are often said to have a relapse, but really they have never stopped having typhoid fever. There is also a considerable number of cases in which the disease only lasts three weeks.

There are cases in which typhoid fever as a disease lasts longer than four weeks, even up to twelve weeks, and during all this time the patients continue to have a temperature.

The cases of post-typhoid sepsis with fever, which will be described later, must be sharply distinguished from these long typhoids.

Complicating inflammations and perforations are accompanied by a rise of temperature; hæmorrhages from the bowels by a considerable fall of temperature.

From time to time we see curious cases in which throughout the disease the temperature runs between 96° and 99° F.

The height of the temperature is regularly in proportion to the severity of the disease, but not always in proportion to the extent of the intestinal lesion.

In the different charts will be found examples of most of the curves of temperature—regular and irregular.

The Pulse.—The ordinary pulse is from 80 to 100 during the first week; more rapid, more feeble, or dicrotic, as the disease goes on.

There are cases in which the pulse is like that of cerebro-spinal meningitis—a slow pulse with a high temperature.

Liebermeister's paper on typhoid fever, published in 1874, has had a profound and unfortunate effect on the mind of the profession as regards the condition of the heart in the disease. He teaches that in cases that run their course without complications the gravest and most frequent danger to the patient is weakness or paralysis of the heart.

This is absolutely untrue. The toxins of pneumonia and diphtheria have a direct effect on the heart, the toxin of typhoid fever has no such effect. The heart may share in the general weakness due to the long continuance of the disease and scanty feeding, but that is all. In ordinary cases, with good nursing and careful feeding, heart failure need not be thought of.

At the end of a typhoid fever, and after it, however, I have seen two serious forms of heart failure.

One seems to be due partly to exhaustion from the long illness, partly to insufficient food. It is not usually fatal, but it can be.

CASE XIV. *Fatal Syncope About the End of the Fourth Week.*

Female, 25 years old; was taken sick about three weeks before admission to the hospital with headache, fever, and prostration.

When admitted she was emaciated and feeble; evidently had been badly cared for; tongue brown and dry, pulse 92, temperature 104°; a few rose-colored spots on abdomen.

The next day she had an attack of syncope, during which the pulse could not be felt, lasting for a few seconds.

The following day she had five such attacks of syncope.

The next day she had several similar, and died in one that evening.

Autopsy.—Brain not examined.

Heart and lungs normal.

Liver normal; spleen large and soft; kidneys firm and congested; stomach thickly coated with mucus.

In the ileum there were a few small ulcers of the solitary and agminated glands.

The mesenteric glands were swollen and infiltrated with pus.

The colon was normal.

The other form is different.

It occurs at the end of the fourth week or later in patients who have been well cared for. It is accompanied with a rise of temperature as if with a fresh sepsis.

One case of this kind I saw in a young man at the end of a rather mild four-weeks' typhoid. For two days he seemed to be dying, with temperatures of 104°, and a heart action so feeble and irregular that it seemed as if he might die at any minute. He recovered, and has remained well for two years.

The other case was a woman, 40 years old, who had been well-nursed and cared for, except that she had not been well fed. She ran a pretty severe typhoid for nine weeks. Then for a week she seemed to be convalescing. At the end of that time the temperature suddenly ran up to 103° to 106°, with very feeble heart action, and in forty-eight hours she was dead.

Autopsy.—There were no lesions of the heart, lungs, liver, or spleen.

There was no peritonitis and no swelling of the mesenteric glands.

In the ileum there were a number of cicatrized ulcers.

The colon was normal.

The epithelial cells of the kidneys were very much degenerated.

Cerebral Symptoms.—Headache, restlessness, irritability, and sleeplessness belong to the first week of the disease.

In the second week the patients are in a condition of apathy.

In the third week are regularly developed alternating delirium and stupor.

A violent delirium may come on in the first days of the disease.

The characteristic apathy may be absent, even in fatal cases.

There are some patients in whom the headache is very severe at first, and in whom there is later developed a stupor which continues until death. These cases resemble cerebro-spinal meningitis.

The eruption is in the form of small, isolated, rose-colored, lenticular spots, slightly elevated, and disappearing on pressure. It appears on the front of the chest and abdomen, sometimes on the back. There are only a few spots, or many. It appears between the seventh and twelfth days, exceptionally between the fourth and twentieth days. There are successive crops of eruption, so that it lasts from seven to twenty-one days, but it may only last from two to four days.

Relapses are marked by a fresh eruption. Spots may continue to appear after convalescence has commenced.

In some cases no eruption can be demonstrated.

The tongue is at first moist, with a broad strip of white fur down the centre. In the milder cases the tongue remains moist throughout the disease, in the more severe cases it becomes dry, brown and fissured. Or the tongue remains clean, but becomes dry, glazed and fissured.

Nausea and vomiting are often present during the first week. Later in the disease there may be vomiting from the tympanites, from the retention of food in the stomach, or from peritonitis.

Some distension of the intestines with gas is usually present; marked distention is an unfavorable symptom; extreme distention may so interfere with the action of the heart and lungs as to cause alarming pulmonary and cardiac symptoms.

CASE XV. *Early and Excessive Tympanites with Great Dyspnœa; Irregular Temperature; Convalescence on Fifteenth Day.*

Male, 15 years old, was suddenly taken sick in the night with vomiting, great pain in the diaphragm, rapid breathing, fever, and prostration.

Second day: The vomiting continues, great pain in the diaphragm, rapid breathing, abdomen hard and tympanitic, sleepless, irritable, fever.

Third day: His condition was the same; the bowels were moved with an enema.

Fourth day: The tympanitis is increasing, the breathing is rapid and labored, the pharynx is inflamed, he coughs up bloody mucus.

Fifth day: Temperature 102° to 99° F., tongue coated, the tympanitic distension of the abdomen is still increasing.

Sixth day: Temperature 101° to 104°, pulse 124, breathing 34.

Seventh day: Temperature 102° to 103°, pulse 112 to 116, breathing 30 to 42. In the afternoon there were several movements from the bowels, the pulse ran up to 140, there was apparent collapse, the nurse thought he was dying.

Eighth day: Temperature 102° all day, pulse 118 to 120, breathing 40 to 42.

Ninth day: Temperature 102° to 99°, pulse 112 to 118, breathing 44 to 54. There was again a movement of the bowels followed by great prostration.

Tenth day: Temperature 99° to 98°, pulse 100, breathing 42 to 50.

After this there was no more fever, but the breathing continued rapid, and he was not convalescent until the fifteenth day.

He was given solid food on the sixteenth day.

He was not able to sit up until the twenty-seventh day.

Diarrhœa is considered one of the regular symptoms of the disease, but in New York it is more often absent than present. There are several loose fæcal passages every day of the appearance of pea soup. Such a diarrhœa may be one of the first symptoms; it may not come on until the third or fourth week; it may not begin until after the patient has taken a purgative; it may last for a few days or throughout the disease. A severe and uncontrollable diarrhœa usually means extensive and deep ulcers.

Bleeding from the intestines is always a serious matter. During the first week there may be moderate bleeding from the congested mucous membrane.

The more important bleedings are from the ulcers in the intestines. They occur most frequently in the third and fourth weeks, but are sometimes seen as early as the second week, sometimes not until after the fourth week, or even in convalescence. The hæmorrhages are small or large, single or repeated. Large hæmorrhages are regularly followed by a temporary fall of temperature. The hæmorrhages are usually, but not always, preceded by diarrhœa. Such hæmorrhages are sometimes fatal, and it is even possible for a patient to bleed to death in this way without the escape of any blood from the rectum.

CASE XVI. (Figure 13.) *Five Weeks' Typhoid; Repeated Bleeding from Intestines; Recovery.*

Male, 20 years old, went to bed five days ago with headache, chilliness, vomiting, diarrhœa, and prostration.

Sixth day: The temperature chart begins; Widal's test positive; eruption well marked. Was ordered tub baths at 70° F.

Twelfth day: Tympanitic and delirious.

Fifteenth day: Three ounces of blood in the stool.

Nineteenth day: Passed two pints of blood at one time from the intestines.

Twenty-first day: Passed two ounces of blood.

Twenty-sixth day: Passed two ounces of blood.

The disease ran its course and terminated in recovery at the end of the fifth week.

CASE XVII. (Figure 14.) *Four Weeks' Typhoid; Repeated Bleedings; Recovery.*

Male, 30 years old, for two days had chills, pharyngitis, and general malaise. On the third day he went to bed; the temperature chart begins on that day.

Ninth day: There was diarrhœa.

Fourteenth day: Bleeding from the bowels, with fall of temperature.

Sixteenth day: Two bleedings, with fall of temperature.

He was convalescent on the thirtieth day.

CASE XVIII. *Fatal Bleeding from the Bowels on About the Seventh Day.*

Male, 22 years old, has had a diarrhœa for a few days without any other symptoms.

First day: He went to bed; temperature 99°, pulse 100, abdominal pain and diarrhœa.

Second day: Temperature 101°, pulse 100, dry tongue, diarrhœa checked.

Fifth day: Temperature 102°, pulse 110.

Sixth day: Temperature 102°, pulse 125, delirious, passed large quantities of blood from the bowels.

Seventh day: He died suddenly.

Autopsy.—The brain was not examined.

The heart was soft.

The lungs were normal.

The liver and spleen were large and soft.

In the mucous membrane of the stomach there were a number of small round ulcers.

In the ileum the agminated and solitary glands were swollen and ulcerated.

The mesenteric glands were swollen.

In the colon, for its entire length, the solitary glands were swollen and ulcerated.

CASE XIX. (Figure 15.) *Four Weeks' Typhoid; Bleeding from Bowels on the Twenty-third Day; Convalescence on the Twenty-ninth Day; Fatal Bleeding on the Thirty-seventh Day.*

Female, 24 years old, about two weeks before coming into the hospital began to be chilly, feverish, with loss of appetite, headache, nausea, and diarrhœa.

Tenth day: Temperature 99° to 102°, headache, diarrhœa, eruption over abdomen, spleen enlarged, does not look very sick.

Twelfth day: The temperature rose to 104°, she became wildly delirious.

Fifteenth day: The temperature chart begins.

Sixteenth day: Less delirious, diarrhœa.

Twentieth day: Comfortable, sleeps well, constipation.

Twenty-second day: More diarrhœa; she is very feeble.

Twenty-third day: A large bleeding from the bowels.

Twenty-fourth day: Diarrhœa, but no more blood.

Twenty-ninth day: She seems to be fairly convalescent.

Thirty-seventh day: In the morning she became very restless and refused food and medicine; there was a large bleeding from the bowels, with a fall of temperature to 96°.

Thirty-ninth day: She died.

In the ileum the agminated glands were changed into clean, smooth ulcers.

The colon was honeycombed with smooth, clean ulcers.

CASE XX. (Figure 16.) *Death on the Thirteenth Day from Bleeding from the Ileum into the Colon, the Blood not Passed During Life.*

Female, 20 years old.

First day: Chill, headache, cough, anorexia, constipation, prostration.

Second day: Vomiting.

Fifth day: Temperature chart begins. The tongue is coated and moist, there is tympanites, there are subcrepitant râles over the lower lobes of both lungs, urine 1032, a little albumin, a few hyaline and granular casts.

Seventh day: Widal test negative, tongue dry, considerable tympanites.

Thirteenth day: Coughing, breathing rapid, apathetic, involuntary urine and fæces.

Twelfth day: In the night the temperature began to rise, the pulse to become rapid and feeble, the patient failed rapidly and died the next morning.

Autopsy.—The brain was not examined. Both lungs showed the lesions of bronchitis and broncho-pneumonia.

The spleen was four times its natural size.

The liver and kidneys showed the changes of moderate acute degeneration.

In the ileum the solitary and agminated glands were swollen and ulcerated.

The colon was nearly full of blood; its glands were not changed.

CASE XXI. *Death from Bleeding on the Thirteenth Day.*

Male, 25 years old, had not been feeling well for about a week. Four days before admission, after working in an ice house, he caught cold and had cough, headache, fever, and sweating.

Now, temperature 103°, face flushed, tongue dry and brown, bowels loose, mind dull and apathetic, urine contained a little albumin. There was a good deal of dyspnœa, with coarse and subcrepitant râles over both lungs. The symptoms of severe bronchitis continued; on the eighth day of his disease he became delirious, the temperature ran from 100° to 103°. On the morning of the twelfth day he had five large passages of pure blood and the temperature fell to 97°. On the thirteenth day the temperature rose to 108° and he died.

Autopsy.—The brain was not examined. The heart was normal.

The lungs were congested and œdematous, the large and medium-sized bronchi were congested and œdematous.

There was a small abscess in the right lobe of the liver.

The spleen was large and firm.

The kidneys looked normal.

In the ileum there were large, clean ulcers, with thick edges of the agminated and solitary glands.

The mesenteric glands were moderately swollen.

The colon was normal.

Some general idea of the bleedings in typhoid can be obtained from the following short notes of 18 cases:

1. Hæmorrhagic typhoid: at onset nose bleed and blood in the stools. In the fourth week, bleeding from the nose, gums, bladder, and intestines. Death in the sixth week.

2. On the tenth day a large bleeding from the bowels, without fall of temperature. On the twelfth day, a small bleeding. Death on the fourteenth day.

3. In the sixth week a constant discharge of blood from the rectum for three days, and then death. The ulcers in the ileum were all clean.

4. During the fifth week, after convalescence had commenced, six large bleedings from the bowels, and death on the same day. There were only small ulcers of a few agminated glands in the ileum.

5. On the ninth day, moderate bleeding, with a fall of temperature. On the tenth day, two bleedings. Death in the tenth week from dysentery.

6. At the end of the fourth week, a large bleeding. At the end of the sixth week a large bleeding and death. The ulcers in the ileum were cicatrized. In the colon there were a number of clean ulcers.

7. On the eleventh day, a number of bleedings, followed by a fall of temperature. Death on the thirteenth day. In the ileum there were large ulcers with thick walls from which the sloughs had cleared off.

8. The fæcal passages were streaked with blood during the first week. On the eleventh day there were large bleedings, followed by death on the next day. The solitary and agminated glands in the ileum were swollen and ulcerated, the solitary glands of the colon were swollen and ulcerated.

9. There were a number of small bloody passages during the fifth week, followed by death. There were large, deep, clean ulcers in the ileum.

10. During the third week there were a number of large, bloody passages. The patient recovered after six weeks.

11. There were three bloody passages at the end of the sixth week; convalescence two weeks later.

12. On the seventeenth day there was a little blood with the passages; convalescence on the twenty-third day.

13. At the end of the fifth week there were a number of small bleedings; convalescence three weeks later.

14. On the tenth day there were large bleedings, followed by a fall of temperature; convalescence in the tenth week.

15. On the seventh day, several bleedings, followed by a fall of temperature; convalescence on the twenty-eighth day.

16. In the seventh week, six large passages of pure blood, followed by a fall of temperature; convalescence in the eleventh week.

17. At the end of two weeks, one bloody passage; at the end of four weeks, four bloody passages; convalescence three weeks later.

18. At the end of two weeks, one bloody passage; at the end of five weeks, two large, bloody passages; convalescence two weeks later.

Abdominal Pain and Tenderness are common, but not necessary symptoms. Louis noted abdominal pain in 106 out of 127 cases, and it is spoken of as of common occurrence by Murchison and others.

In some cases there are definite reasons for the pain and tenderness—diaphragmatic pleurisy, inflammation of the gall bladder, tympanitic distension of the intestine, inflammation of the appendix, perforation and peritonitis, either local or general. The typhoid ulcers do not seem to give pain, unless there is bleeding.

But there remains a large number of cases where there is pain in different parts of the abdomen without discoverable cause. These pains, especially if there is also increased leucocytosis, lead to errors in diagnosis. Dr. M'Crea (*N. Y. Med. Jour.*, Mar. 4, 1901), has given an excellent account of some of these cases.

He found in 70 cases abdominal pain without discoverable cause. In 14 of the cases the leucocytosis was from 10,000 to 17,000. In two of the cases the abdomen was opened and nothing was found.

Epistaxis is very often present. It occurs at the onset of the disease, or at any time in its course. It may occur only once, or be repeated; it is seldom so profuse as to be dangerous.

The eyes in the third week may become somewhat insensible to light, with dilated pupils.

Ringing and buzzing sounds in the ears may be complained of during the first week; in the third or fourth week there may be more or less deafness.

Catarrhal pharyngitis is present in nearly every case. It may hardly be noticed, or give pain, or a troublesome cough.

Streptococcus and staphylococcus inflammations of the mouth and throat ought not to occur with care and good nursing, but yet they sometimes do.

CASE XXII. *Staphylococcus Sore Mouth on the Sixteenth Day of a Four Weeks' Typhoid.*

Female, 18 years old, was taken sick in the country. On the sixth day she was brought to the city and went through the rest of her illness in a new, large, clean, well-ventilated house on Riverside Drive.

The typhoid was a severe one, the temperature at or above 104°, some delirium, but very little emaciation. She was treated with warm tubs. Convalescence began on the twenty-eighth day.

Sixteenth day: The lower lip was swollen at the right corner of the mouth.

Seventeenth day: There was a patch of dirty false membrane on the inside of the lower lip extending backward. A boro-formalin mouth wash was used.

Eighteenth day: The lip was less swollen and the membrane smaller, but in her delirium she bites and picks at it.

Twenty-second day: The lip had been doing well until in the night she chewed away a piece of the inside of the cheek at the left angle of the mouth. This was soon covered with a false membrane and there was more membrane at the right angle of the mouth. She was now given douches of bichloride of mercury; she was fed with rice once a day in addition to the milk.

Twenty-fifth day: The false membrane is now extending so as to cover most of the inside of the lips. The irrigations were changed to chloride of zinc and a spray of argyrol was also used. She was given chicken to eat.

Twenty-sixth day: The false membrane has gone and the ulcers are clean.

Twenty-seventh day: She sits up in a chain.

Thirty-fourth day: The ulcers are entirely healed.

Catarrhal or croupous laryngitis, ulcers of the larynx, and œdema of the glottis are occasionally met with.

Suppurative inflammation of the parotid glands used to be not infrequent; now, with good nursing and cleanliness, it is seldom seen.

Bronchitis may be developed at any time in the disease. It is usually confined to the larger tubes and is not very severe; but there are cases of purulent bronchitis that are very serious.

Broncho-pneumonia is a much more serious complication. It may be developed at any time in the course of the disease. When it occurs in the first week it is difficult to tell whether the patients have pneumonia or typhoid fever with pneumonia.

CASE XXIII. *Broncho-Pneumonia with Typhoid Fever; Death on the Seventh Day.*

Male, 21 years old, seven days before death began to have diarrhœa, but continued to work for two days.

On the sixth day of his illness he came into the hospital. He was cyanotic, delirious, tongue dry and coated, temperature 104°, pulse 132, breathing 42.

Seventh day: Temperature 104°, pulse 124, breathing 64; several dark fluid passages from the bowels; he died.

Autopsy.—The brain was not examined.

The heart was covered with old pericardial adhesions, the muscle was firm.

There was fibrin on the pulmonary pleura. The lungs were congested. The bronchi were congested, the larger ones coated with mucus, the smaller ones containing pus. The walls of the smaller bronchi were swollen and infiltrated with cells. Around these small bronchi are zones of congestion, or pneumonia.

Lobar pneumonia and typhoid fever may both be present at the same time. The combination seems to be an accidental one. The symptoms of either disease may predominate.

CASE XXIV. *Typhoid Fever; Lobar Pneumonia; Death.*

Male, 24 years old, nineteen days before death was said to have taken cold and to have had a cough.

Twelve days ago he went to bed with cough, headache, chills, fever; soon became delirious, and later had diarrhœa.

Fifteenth day: Delirious, face flushed, tongue coated, eruption on abdomen, tympanitic, diarrhœa, physical signs of bronchitis.

The temperature ran from 102° to 104°, the pulse from 116 to 170, the breathing from 30 to 40.

On the nineteenth day he died.

Autopsy.—The brain was not examined.

The lower lobe of the right lung was in the condition of red hepatization, the rest of the lung was congested.

The heart, liver and kidneys were normal. The spleen was large and soft.

The mesenteric glands were swollen.

In the ileum the solitary and agminated glands were ulcerated and swollen.

The Blood.—During the course of typhoid fever a moderate secondary anæmia is regularly developed, the red cells fall to about 4,000,000 and the hæmoglobin to 70 per cent. Sometimes, especially with bleedings from the bowels, the red cells fall as low as 2,000,000. In convalescence the secondary anæmia is often fairly well marked and may require the use of iron.

The white cells are regularly diminished in typhoid fever, and this is of some value in diagnosis. An increased leucocytosis regularly means the presence of a complicating inflammation. Unfortunately there is a considerable number of cases in which the leucocytes are largely increased for a time without discoverable cause.

The urine varies with the condition of the kidneys and that of the bladder. The presence of typhoid bacilli in the urine constitutes an irritant capable of setting up a cystitis. As a matter of fact, however, we do not often see cystitis unless a catheter has been passed, or the bladder has been infected by some other form of bacteria.

The most common lesion of the kidneys is a mild form of acute degeneration with a little albumin and a few casts in the urine. In the protracted cases, however, the degeneration of the epithelium becomes very marked. Less frequently there is an acute exudative nephritis with diminution in the quantity of urine, numerous casts, and considerable quantities of albumin. Such an acute nephritis may occur at the very beginning of a typhoid.

Thrombosis of the veins belongs to the third and fourth weeks of typhoid fever; it occurs quite frequently.

The femoral and external iliac veins are the ones most frequently affected. The obstruction of the vein by the thrombus causes swelling and œdema, the inflammation of the vein causes pain, sometimes very severe. In addition, at the time of the thrombosis there may be a rise of temperature and evidences of fresh infection. The condition is not likely to prove fatal, but the affected leg may be left swollen and painful for many months.

CASES LXX., LXXI., LXXII., LXXIV.

With thrombosis of the sinuses of the dura mater there is hemiplegia, loss of consciousness, delirium, and a rise of temperature. The condition is usually fatal, but recovery is possible.

CASE XXV. *Typhoid Fever; Hemiplegia; Recovery.*

Male, 29 years old, November 5, 1883.

Eleven weeks before admission he was taken sick with headache, diarrhœa, and irregular fever; after three weeks he went into a hospital in Chicago and was treated for typhoid fever for six weeks. He left Chicago, although very weak, came to New York, and entered the hospital here.

Now, temperature 104°, pulse full and rapid, mild delirium, tongue dry and brown, abdomen tympanitic, typhoid eruption, diarrhœa.

November 13: The temperatures have run between 101° and 104°, but to-day between 100° and 101°, pulse good, tongue is clearing off, still has diarrhœa, is dull and stupid.

November 14: There was suddenly developed complete paralysis of the left face, arm and leg, dull and stupid, tongue brown and dry, temperature 104°.

November 15: Temperature 98°.

November 17: Temperature 99°, tongue dry and brown, is dull and stupid, complete paralysis of left side.

November 19: The temperature continues normal, but he is in the condition of alternating delirium and stupor, with brown, dry tongue. He can move the left leg a little.

November 23: He is still delirious, but can eat, and moves the left leg.

November 27: He is still delirious, but has more power in the left leg.

After this he gradually improved, and by December 31 was quite well, and had almost entirely regained power on the left side.

CASE LXXIV.

Thrombosis of the ventricles of the heart causing death I think I have seen in one case. (Heart, page 357.)

Perforation of the wall of ulcers in the small intestine, appendix, and colon now constitutes one of the greatest dangers of typhoid fever. It occurs both in mild and severe cases at any time after the ulcers are formed until they are entirely healed; that is, from the end of the first week into convalescence. The perforation causes a certain amount of shock and pain and is at once followed by peritonitis.

In a fair number of cases of perforation the symptoms are so characteristic that the diagnosis is easy. First, the sudden collapse of the patient with abdominal pain, vomiting, rise of temperature, rapid and feeble pulse. Then the symptoms of acute peritonitis: pain, tenderness, rigidity of the abdominal wall, vomiting of brown fluid, high temperature, rapid pulse, increased leucocytosis.

But, unfortunately, a number of cases are very obscure. There is nothing to show the existence of perforation or peritonitis. The patients simply at some time seem to be sicker than they were before with their typhoid.

Still further than this it is now established that abdominal pain and increased leucocytosis can be present without perforation or peritonitis.

CASE XXVI. *Perforation and Peritonitis.*

Male, 28 years old, September 19, 1883. Five days ago he began to feel tired, weak, with vomiting and abdominal pain.

Three days ago he began to have diarrhœa.

Now, temperature 102°, tongue moist and coated, typhoid eruption, diarrhœa, delirious, urine 1014, albumin, granular casts.

The temperature ran along at about 100° until September 23, when it fell to 99° and he developed the symptoms of general peritonitis.

September 25: The temperature rose to 106° and he died.

Autopsy.—The brain was not examined. The walls of the heart were soft and the muscle pale.

The lungs were congested.

The liver was large and pale.

The stomach was normal.

The peritoneum was coated with fibrin.

In the lower part of the ileum there was partial ulceration of the agminated glands, the ulcers nearly cleared off. One small ulcer had perforated.

The mesenteric glands were a little swollen.

The colon was normal.

The kidneys looked normal.

CASE XXVII. *Perforation During Convalescence.*

Male, 24 years old, September 1, 1881.

August 27: He began to have headache, abdominal pain and fever.

Now, pulse 92, temperature 102°, bloody sputum, sibilant and sonorous râles over both lungs.

He went through the course of a moderate typhoid fever and on September 21 was convalescent.

October 9: He was out of bed.

October 27: He left the hospital and went on a spree.

October 29: He came back with the symptoms of general peritonitis.

November 7: He died.

Autopsy.—The brain was not examined.

The heart was normal.

The lungs were congested.

The liver and spleen were large and soft.

The cortex of the kidneys was pale.

The peritoneum was congested and coated with fibrin and pus.

In the upper part of the ileum one small ulcer had perforated. All the other ulcers were healed.

CASE XXVIII. *Perforation of the Appendix After Typhoid Fever.*

Male, 30 years old, May 16, 1876.

Three weeks ago he began to have headache, nausea, general pains and fever. He went into a hospital, was treated for a week, and then discharged. He continued to have fever and feel sick.

May 16: He has a temperature of 105°, brown, dry tongue, delirium and tympanites. Neither perforation nor peritonitis were recognized.

He continued to have temperatures between 101° and 104°, with occasional chills, and died on May 29.

Autopsy.—There was an increase of serum beneath the pia mater.

The heart, liver, spleen, stomach and kidneys were normal.

The lungs were congested.

In the lower part of the ileum there were deep, clean ulcers of the agminated glands.

In the vermiform appendix there was a deep typhoid ulcer which had perforated.

The mesenteric glands were enlarged and suppurating.

The peritoneum was coated with fibrin.

Peritonitis is produced, not only by perforation, but also by an extension of the inflammation from the wall of the intestines, by inflammation of the mesenteric glands, and by infarctions of the spleen. It usually gives well-marked symptoms of acute general peritonitis, and is very fatal.

CASE XXIX. *Peritonitis During Convalescence Due to an Inflamed Mesenteric Gland.*

Female, 20 years old, July 20, 1877.

About July 5 she began to feel chilly, have headache, backache, and diarrhœa.

July 12: She went to bed with vomiting and delirium.

July 20: The tongue is dry, typhoid eruption, diarrhœa, tympanites, large spleen, delirium, urine 1009, a little albumin.

She continued very sick until July 30, when she began to improve.

August 1: She was worse, with dulness and subcrepitant râles over the lower lobe of the right lung.

August 4: The diarrhœa has stopped, the temperature is normal, she is sitting up, convalescent.

August 13: She began to have severe pain in the back, rapid pulse, constant vomiting, great prostration, and died August 16.

Autopsy.—The brain was not examined.

The heart was normal.

The lower lobe of the right lung was congested and œdematous.

The liver and spleen were normal. The kidneys showed the changes of acute degeneration.

In the ileum all the agminated glands were in the condition of clean cicatrizing ulcers.

The mesenteric glands are swollen, the peritoneum over them is congested and coated with fibrin.

The Colon may exhibit the same changes in its solitary glands that we find regularly in the glands of the ileum, and sometimes the lesion in the colon is more marked than that in the ileum.

This seems to make no difference in the symptoms, which are those of an ordinary severe typhoid.

I have seen a number of cases of inflammation of the lower end of the colon after typhoid fever. The clinical picture is that of a croupous colitis: frequent small passages of blood and mucus, a febrile movement and considerable prostration.

The anatomical picture is that of false membrane and necrosis.

It has seemed probable to me that the condition is due to a streptococcus infection from the tubes used to give enemata.

CASE XXX.—*Acute Colitis After Typhoid Fever; Recovery.*

Male, 16 years old, August 28, 1882.

Three weeks ago began to have headaches and general malaise, with constipation.

One week ago felt so badly that he went to bed.

August 28: Temperature 105°, tongue dry and brown, bowels constipated, no eruption.

He ran the course of an ordinary typhoid with persistent constipation, for which he was given laxatives and enemata.

The temperature fell to the normal and he was convalescent on September 13, but continued to receive enemata.

September 30: He left the hospital. Twenty-four hours after leaving the hospital he began to have small, frequent, painful passages of blood and mucus.

October 5: He was readmitted. Temperature 101°, tongue dry; small, frequent, painful passages of blood and mucus.

This condition continued until October 19, when the mucous and bloody passages ceased and he began to have natural fæcal passages.

CASE XXXI. *Acute Colitis After Typhoid Fever; Recovery.*

Male, 22 years old, August 22, 1882.

About ten days ago he began to have vomiting, general malaise, sore throat, and fever.

August 22: Temperature 105°, no eruption, constipated.

He was treated with cold tubs, laxatives, and enemata.

September 27: The temperature fell to the normal and convalescence began.

September 28: He began to have abdominal pain and diarrhœa, with a good deal of prostration.

September 29: He had small, frequent, painful passages of blood and mucus.

This condition continued until October 8, when he began to improve, and went on to complete recovery.

Case XXXII. (Figure 17.) *Four Weeks' Typhoid; Irregular Temperature; Bleeding from the Intestines; Treatment by Cold Tubs; Post-Typhoid Sepsis; Croupous Colitis; Death.*

Male, 21 years old; two days ago had a chill.

Third day: The chart begins. He does not look very sick; has a good deal of pharyngitis.

Ninth day: Vomiting and diarrhœa.

Fourteenth day: Bleeding from the bowels.

Twenty-eighth day: Temperature normal, convalescent.

From the forty-second until the seventieth day he was in the condition of post-typhoid sepsis. He was not allowed out of bed until the sixty-fourth day.

Sixty-sixth day: He began to have small, frequent, painful passages of blood and mucus. These continued until he died on the seventy-sixth day.

Autopsy.—The brain was not examined.

In the upper lobe of the left lung there were miliary tubercles and small, cheesy foci. The bronchial glands were suppurating.

The heart, liver and spleen were small.

The kidneys looked normal.

The mesenteric glands were swollen. In the lower part of the ileum a number of the agminated glands were replaced by small, shallow, cicatrized ulcers.

The lower two-thirds of the colon was coated with a uniform, thick layer of fibrin; under this there was necrosis of the mucous membrane.

Case XXXIII. *Amœbic Colitis After Typhoid Fever; Death.*

Male, 22 years old, August 29, 1873.

For two weeks he has had general malaise, diarrhœa, and increasing prostration.

August 26: He went to bed.

After entering the hospital he went through the typical course of a typhoid fever.

September 13: Temperature 98° to 103°.

After this the temperature rose, 102° to 105°, and continued high up to death.

Baths reduced the temperature to 98°, but in three hours it would go up to 104° or 105°.

The bowels were loose from the day of admission, and during the last ten days of his life he had from six to ten watery, yellow stools daily.

For the last five days there was vomiting, the mind remained clear, he became extremely emaciated. Just before death the temperature was 105°, pulse 168, breathing 60.

October 10: He died.

Autopsy.—The lungs and heart were normal.

The liver was congested.

The kidneys showed the changes of acute degeneration.

The spleen was large and firm.

The mesenteric glands were enormously swollen.

In the lower part of the ileum there were two typhoid ulcers of agminated glands extending down to the muscular coat and cleaned off.

The upper two-thirds of the colon was thickly studded with small and large ulcers. These ulcers have the appearance of amœbic ulcers, with thick, overhanging edges. There are amœbæ in their walls and floors.

Jaundice.—Catarrhal gastritis is of such ordinary occurrence in the first weeks of typhoid that one would look for a number of cases of gastro-duodenal jaundice, but as a matter of fact it is not very often seen.

CASE XXXIV. *Typhoid Fever with Jaundice.*

Male, 25 years old.

Fifteen days before admission loss of energy, drowsy.

Eight days before admission headache, nausea, vertigo, sleeplessness.

Five days ago went to bed and after that had nose bleed every day. On the day before admission became jaundiced.

Sixth day of disease admitted to the hospital. He is prostrated, jaundiced, and tongue red and dry, no vomiting, temperature 101°, urine 1014, albumin, bile, hyaline and granular casts.

From the sixth to the twenty-first day the morning temperatures were about 100°, the afternoon temperatures 102.6°.

Until the twenty-seventh day the jaundice continued with nose bleed, itching, petechial spots in the skin, and clay-colored stools.

There was no Widal reaction on the fifteenth and twenty-fourth days.

He was convalescent on the twenty-seventh day and out of bed on the thirtieth day.

INFLAMMATION OF THE GALL BLADDER.

This condition occurs during convalescence. During the course of any typhoid the characteristic bacilli are regularly present in the bile within the gall bladder. But it is only in a moderate number of cases, and that during convalescence, that a sero-purulent inflammation of the wall of the gall bladder is developed. This seems to indicate an infection with some additional irritant.

The gall bladder becomes distended and contains bile, mucus, serum, pus, and sometimes blood. The mucous membrane is congested and swollen. The peritoneal surface may be coated with fibrin.

Some of the cases have the ordinary symptoms of an acute purulent inflammation of the gall bladder. The distended gall bladder can be felt; it is tender and painful. The constitutional symptoms are those of sepsis: nausea, vomiting, a rapid pulse, an irregular temperature, marked prostration.

In other cases there is nothing to call attention to the gall bladder, but yet it is evident that something is interfering with convalescence.

It is said that in a considerable number of persons gall stones are developed after typhoid fever.

I have seen one curious case of obstructive jaundice occurring a year after typhoid fever. The patient went through an ordinary four weeks' typhoid, but convalescence was slow and interrupted by irregular rises of temperature. Finally he recovered and was entirely well for about one year. Then without any other symptoms he suddenly became jaundiced. The jaundice continued and deepened for five weeks, then the abdomen was opened. It was found that the gall bladder was contracted and covered by adhesions. These adhesions constricted the common duct. This condition was relieved by operation, and he has been very well for a year.

Inflammations of the epididymis and testicle are seen in a moderate number of cases. They may occur during the course of typhoid fever, but belong rather to the period of convalescence. They are usually unilateral, but may be bilateral, and involve either the epididymis or testicle, or both. Usually there is only congestion and swelling, terminating in resolution, but there may be suppuration or necrosis.

CASE XXXV. *Epididymitis and Orchitis on the Nineteenth Day of Typhoid Fever.*

Male, 23 years old, admitted to the hospital on the seventh day of his typhoid.

He ran a temperature of 99° to 103° till the seventeenth day; then there was a fall of temperature and subsidence of symptoms till the twenty-fifth day. Then the temperatures ran from 102° to 104° until the thirty-ninth day. Convalescence was not established until the forty-second day.

Inflammation with pain and swelling of the left epididymis and testicle began on the nineteenth day and lasted for about two weeks.

Inflammations of the periosteum and joints belong to the period of convalescence, but may occur during the typhoid fever. They are caused by the presence of typhoid bacilli in the inflamed tissue. The inflammation may only cause swelling or go on to suppuration; with periostitis there may be necrosis of the underlying bone. The inflammation is regularly sluggish, long continued, and prone to relapse. The tibiæ and ribs are the bones most frequently involved.

CASE XXXVI. *Periostitis on the Twelfth Day of Typhoid Fever.*

Male, 32 years old, nine days before admission began to feel sick with chilliness, prostration, and diarrhœa. Two days ago he vomited and went to bed. Third day of typhoid, temperature 106°, pulse 96.

He was treated with cold tubs from the fourth to the thirteenth day.

Twelfth day: The left external malleolus was red, swollen, œdematous, and tender. The next day the swelling extended up the leg.

Twenty-first day: The swelling was opened and two ounces of pus removed.

Twenty-third day: There was a red, tender swelling between the thumb and finger of the left hand.

The next day this was opened and one ounce of pus evacuated.

Thirty-ninth day: Both wounds have healed.

CASE XXXVII. *Periostitis During Convalescence.*

Male, 28 years old, June 25, 1900, was admitted on the eighth day of his typhoid. He went through an ordinary typhoid and was convalescent on the twenty-ninth day.

He left the hospital on the forty-third day. Within a few days after leaving the hospital a lump appeared over the fourth left costal cartilage, and gradually increased in size until it was as large as a pigeon's egg; there was a little redness and marked pain and tenderness.

The swelling, pain, and tenderness continued, and in October, 1900, it was aspirated, but no fluid was found.

After this the tumor was strapped and became smaller and less painful.

The swelling and pain did not entirely disappear until the summer of 1904.

Case XXXVIII. *Arthritis of Wrist Joint During Convalescence.*

Female, 25 years old, came into the hospital on the seventh day of her typhoid fever.

She was treated with cold tubs until the thirty-fifty day.

She developed a cystitis on the fifteenth day.

The fever ran a fairly severe course, without complications, and terminated in convalescence on the forty-fourth day.

She began to have pain in the right wrist on the thirty-ninth day. On the seventy-second day the wrist was aspirated, seropurulent fluid was drawn off from which a pure culture of typhoid bacilli was made. On the seventy-fourth day the wrist was opened, and after that she made a good recovery.

The curious condition called the typhoid spine seems to be either due to periostitis of the vertebræ, or to be an hysterical neurosis. A very excellent analysis of twenty-six cases is given by Dr. F. I. Lord (*Boston Med. and Surg. Journal*, 1902).

The time of onset varies from the latter part of the typhoid to three months after defervescence. The pain is very severe, situated in the lower dorsal or lumbar region, radiating downward or forward. There are regularly remissions and exacerbations, the latter brought on by motion. There may be very little pain, except on motion. The pain is the principal symptom, but there may be added a number of hysterical phenomena. The average duration is about four months.

IRREGULAR CASES.

Cases of Two Weeks' Duration.

In quite a number of cases typhoid fever runs its course and terminates in convalescence at the end of two weeks. Very often this short duration is ascribed to some particular form of treatment, but it seems really to be the natural course of some of the cases.

CASE XXXIX. (Figure 18.) *Two Weeks' Case; No Treatment.*

Male, 19 years old, three days before admission began to have weakness, vertigo, headache, sleeplessness, nose bleed, nausea, fever and sweating.

Fourth day: Tongue coated, urine 1024, no albumin, no diarrhœa.

Sixth day: Nose bleed, well-marked typhoid eruption. He continued to have the ordinary appearance of typhoid fever, without diarrhœa, until the eleventh day; then the morning temperatures began to fall, sometimes below the normal, and by the fifteenth day he was convalescent. There was no treatment.

CASE XL. (Figure 19.) *Two Weeks' Case; Irregular Temperature; Bleeding from the Bowels; No Treatment.*

Male, 18 years old, three days before admission began to have weakness, dizziness, headache, epistaxis, pharyngitis, diarrhœa, and fever.

Fourth day: Tongue coated and brown, diarrhœa, uvula and soft palate swollen, congested and covered with white exudate.

Sixth day: Alternating delirium and stupor.

Seventh day: Typhoid eruption, tympanites.

Eighth day: Two bloody passages from the bowels.

Fifteenth day: The diarrhœa has stopped and he has become rational.

Seventeenth day: He is convalescent. There has been no treatment except a little quinine and whiskey.

CASE XLI. (Figure 20.) *Two Weeks' Typhoid; Woodbridge Treatment.*

Male, 18 years old, for four days before admission headache, chilly, epistaxis, diarrhœa. For two days in bed.

Third day: Face flushed, tongue coated, typhoid face, tympanites.

Fourth day: Was put on the Woodbridge treatment, which was continued until the twelfth day.

On the ninth day he had a sore mouth.

On the fourteenth day he was convalescent.

The Protracted Cases.

There are cases of typhoid fever that continue long over the regular four weeks. It is of the utmost importance to distinguish these cases from the cases of post-typhoid sepsis. This is not always easy.

CASE XLII. (Figure 21.) *Six Weeks' Typhoid; Chart from the Fourth to the Forty-third Day; No Treatment; Recovery.*

Female, 11 years old, one week before admission headache; three days before admission vomiting, nose bleed, fever, and sweating.

Fourth day: Tongue coated and dry, skin hot.

She went through the ordinary course of typhoid fever, except for its duration. There was a little diarrhœa off and on. There was no treatment.

CASE XLIII. (Figure 22.) *Eleven Weeks' Typhoid; Treatment by Cold Tubs; Period of Improvement in the Sixth Week; Recovery.*

Female, 26 years old, seven days before admission had a chill, fever, vomiting, headache, diarrhœa, and cough.

Eighth day: Tongue moist and coated, pulse 96, a little tympanites, the heart, lungs and spleen give no physical signs of disease, urine 1032, a little albumin, a few granular casts. She was put on the use of cold tub baths.

Sixteenth day: There was a little blood in the stools; the cold tubs were stopped and not resumed.

She continued in the typhoid state until the twenty-ninth day, when she began to improve.

Thirty-sixth day: She was given scraped beef.

Forty-second day: She was out of bed.

Forty-fifth day: Back in bed; milk diet.

Fifty-second and third days: She had chills and vomiting.

Fifty-fourth day: Comfortable, tongue clean and dry; was again given scraped beef.

She improved steadily and was out of bed again on the seventy-seventh day.

There are cases in which the morning temperature falls so low—

to or below the normal—that they may be mistaken for cases of malarial fever. But it is only one of the regular varieties of temperature, and makes no particular difference in the course of the typhoid.

CASE XLIV. (Figure 23.) *Typhoid Fever with Low Morning Temperatures; Cold Tubs for Two Days.*

Male, 25 years old, seven days before admission taken with headache, vomiting, nose bleed, and at once went to bed.

Eighth day: Tongue coated and moist, typhoid eruption, spleen enlarged, no diarrhœa, urine normal.

Ninth and tenth days: He was treated with cold tubs.

Fourteenth day: The Widal reaction is positive.

He was convalescent on the twenty-ninth day. Except for the cold tubs on the ninth and tenth days, nothing was done to affect the temperature.

Relapses.—A true relapse in typhoid fever is a fresh attack of the disease produced by a fresh infection from the patient himself. Such a relapse is attended with a new eruption and a repetition of the symptoms of the disease. The relapse may be more severe than the original attack, it may be fatal.

The intermission between the end of the original attack and the beginning of the relapse is from one to three weeks. During the intermission the patient is frankly convalescent. The tongue is clean, the mind is clear, they sleep at night, they are hungry, they may be out of bed, they may be out of doors.

The relapse lasts from seven to thirty-nine days, the ordinary duration is from ten to fourteen days. It is like a fresh attack of typhoid fever of greater or less severity.

There may be two relapses with two periods of intermission.

Unfortunately patients are often said to have relapses with falls and rises of temperature which are of very different character.

It is a regular feature of the typhoid temperature that there is a temporary fall at the end of the second, third, and fourth week. This regular fall may be exaggerated so that for several days the temperature is normal and the patients are better. But yet if the patient is looked at, as well as the temperature chart, it is evident that typhoid as a disease is still going on. It is a period of improvement, and not a true intermission.

Case XLV. (Figure 24.) *Four Weeks' Typhoid; Temporary Fall of Temperature while the Typhoid was Going On.*

Female, 7 years old, three days before admission began to have fever and headache.

Fourth day: Apathetic, drowsy, tongue coated and moist, urine 1028, trace of albumin, treated with sponge baths.

Seventh day: Alternating crying and stupor, diarrhœa.

The diarrhœa, stupor, and crying continued until the twenty-sixth day. After that she improved and was convalescent on the thirty-first day.

The cases of post-typhoid sepsis, which will be described later, may also be taken for true relapses.

Case XLVI. (Figure 25.) *True Relapse; Four Weeks' Typhoid; One Week Intermission; Three Weeks' Relapse.*

Male, 21 years old, went through a pretty severe typhoid for four weeks. During the third week the temperature was reduced by large doses of antipyrine. He was fairly convalescent for a week. Then the temperature rose gradually from day to day and he had a fresh typhoid lasting for three weeks, terminating in convalescence.

Case XLVII. (Figure 26.) *Two Weeks' Typhoid; Two Weeks Intermission; Two Weeks' Relapse; Temperature Chart from the Second Day.*

Male, 23 years old, had a mild typhoid for two weeks. Then he was convalescent and out of bed for two weeks. Then there was a fresh typhoid, more severe than the first attack. This lasted for two weeks and ended in convalescence.

Case XLVIII. (Figure 27.) *Double Relapse; Typhoid for Eighteen Days; Intermission for Twenty-three Days; Typhoid for Seven Days; Intermission for Eleven Days; Typhoid for Seven Days; Convalescence.*

Female, 12 years old, came into the hospital on the sixth day of her typhoid. She was quite sick, with dry tongue, tympanites, delirium, and a characteristic eruption. She was treated with cold tubs.

On the nineteenth day she was convalescent.

On the thirty-fourth day she was discharged from the hospital.

On the fourtieth day she felt sick again and came back to the hospital.

She was moderately sick for a week. Then she was again convalescent until the sixtieth day.

Then the fever came on again for a week; after that there was permanent convalescence.

Walking Typhoid.—This name is given to the cases which continue to be up and about until a few days before their death. There seems to be nothing peculiar about the typhoid. The curious thing is that patients who are so seriously ill can continue to be up and about.

CASE XLIX. *Walking Typhoid Fever.*

Male, 37 years old, was well until twenty-three days before his death. Then after exposure he began to have bronchitis. He was up and about, but complained of irregular chilly and feverish feelings. He continued to feel miserable but did not go to bed, and five days before death was out of doors with a temperature of 101° to 102°.

Four days before death he was delirious and went half-dressed into the street; temperature 104°.

Three days before death he was in bed with alternating delirium and stupor, diarrhœa and typhoid eruption.

Two days before death, face dusky, tongue dry, delirious, typhoid eruption, temperature 103°.

One day before death, temperature 102° to 105°.

On morning of day of death, temperature 108°; he was bathed with ice water and the temperature fell to 95°. That afternoon he died.

Autopsy.—The brain was not examined.

The heart was normal.

The lungs were congested.

The liver and spleen were congested.

The kidneys looked normal.

The mesenteric glands were swollen.

In the ileum the agminated and solitary glands were enormously swollen, with a few small points of ulceration.

The colon was normal except for some swelling of the solitary follicles in the caput coli.

CASE L. *Walking Typhoid Fever.*

Female, 27 years old.

Two weeks before admission to the hospital she began to have headache, abdominal pains, prostration, vomiting, but kept up and about her work until the day before admission, when she became delirious.

Now, temperature 96.4°, pulse 104, tongue dry and coated, marked prostration, no tympanites, urine 1020, albumin and casts. She died the next morning.

Autopsy.—The brain was normal.

The muscle of the heart wall was degenerated.

The lower lobes of the lungs were congested.

The liver and kidneys showed the changes of acute degeneration.

The spleen was not enlarged, but soft and congested.

In the ileum the agminated and solitary glands were swollen, with superficial necrosis.

Hæmorrhagic Typhoid Fever.—It belongs to many of the infectious diseases that in some of the cases there is a disposition to bleeding. This regularly indicates an unusual virulence of the poison. Such a virulent infection with bleeding is seen in some cases of typhoid.

CASE LI. (Figure 28.) *Hæmorrhagic Typhoid Fever; Bleeding from the Nose, Gums, Stomach, Kidneys, Intestines, and in the Skin; Death on the Forty-first day.*

Male, 31 years old, three weeks before admission began to have a bloody diarrhœa, abdominal pain, headache, epistaxis, and after a week delirium.

Twenty-second day: Emaciated, delirious, abdominal pain, bloody passages from the bowels, tympanites, sibilant râles over both lungs, tongue dry and cracked, hæmorrhagic spots in the skin.

Twenty-fourth day: delirium alternating with stupor; the diarrhœa has stopped.

Thirtieth day: He seems to be better.

Thirty-second day: He is worse, more delirious, but no diarrhœa.

Thirty-seventh day: Bleeding from the nose and gums, more bleeding in the skin.

Thirty-ninth day: Bleeding from the nose and rectum, blood in the urine.

Forty-first day: He died.

Autopsy.—The body was emaciated, the skin was studded with hæmorrhagic spots.

The brain was normal.

The lungs were normal, except for old pleuritic adhesions.

The heart was normal, except for a little old thickening of the aortic and mitral valves.

The mucous membrane of the stomach was mottled with small hæmorrhages. The liver and spleen were large and soft.

The kidneys showed the lesions of acute degeneration, with blood in the pelves.

In the ileum there were large, deep ulcers of the agminated glands.

The mesenteric glands were swollen.

The colon contained a large quantity of clotted blood. The upper two-thirds was studded with large, deep ulcers, in many of which there were blood clots.

AFEBRILE TYPHOID FEVER.

Typhoid fever may run its entire course with evening temperatures of not over 99°, and morning temperatures below 98°, and yet it is evident that the patients are really ill. They are very weak, with feeble heart action.

In 1870 there was an epidemic of typhoid fever in the German army around Paris. The cases were characterized by an abundant eruption, great prostration, violent delirium, marked stupor, but slight abdominal symptoms, and temperatures not over 100°, or not above 98°. The disease was moderately fatal, and after death the ordinary lesions were found.

TYPHOID FEVER COMPLICATED BY MALARIAL POISONING.

A double infection with the organisms of both these diseases is not uncommon, but yet both sets of symptoms do not usually go on at the same time. The malarial symptoms are evident before and after typhoid fever, but not while that disease is running its course.

In temperate climates there is usually no great difficulty in distinguishing between typhoid and malarial fevers. Most of the continued fevers are due to the typhoid poison.

In tropical countries, however, where severe, malarial continued fevers prevail, it is often extremely difficult to determine whether the patient has malaria alone, or typhoid alone, or both together. This difficulty is rendered greater by the fact that in a patient poisoned both by malaria and typhoid, while the typhoid is active no malarial organisms are to be found in the blood from the finger or ear. Still further than this, in bad malarial fevers, although organisms are constant in the blood, they are not always to be found in the blood taken from the skin. It is also to be noted that in armies where the sanitary conditions are bad, diarrhœa and starvation confuse the clinical picture.

CASE LII. *Malarial Fever Followed by Typhoid Fever.*

Male, 32 years old, soldier. One month ago, while in Cuba, began to have chills and fever, which were repeated every day.

Seven days before death he was admitted to the hospital at Montauk with pain in the back and limbs, vomiting, diarrhœa, tympanites, slight jaundice, temperature 101°.

He continued to have vomiting and diarrhœa, became delirious, temperatures 98.6° to 100.4° until he died.

Examination of the blood by Dr. Ewing gave no malarial organisms, increased leucocytosis, positive Widal reaction.

Autopsy.—The brain was not examined.

The lower lobes of both lungs were congested and œdematous, with commencing red hepatization.

The heart was normal.

The liver looked normal.

The spleen was large and soft.

The mesenteric glands were swollen.

In the lower end of the ileum the agminated glands were swollen, with superficial necrosis.

The stomach and duodenum were congested and coated with mucus.

The lower part of the colon was congested and coated with patches of fibrin.

CASE LIII. *Malarial Fever, or Typhoid Fever; Death; No Lesions.*

Male, 25 years old, soldier, admitted to the hospital at Montauk nine days before his death.

He began to have chills and fever while in Cuba, and continued to have them daily up to the time of admission. After admission he looked like typhoid fever, no organisms in the blood, positive Widal reaction.

Temperatures: 101.6°, 99.6°—104°, 104.6°—104.4°, 103.8°—103.6°, 102°—104°, 102°—102.4°, 99.6°—101.4°, 99°—101.6°, 101°.

Autopsy.—The brain was not examined.

The heart and lungs were normal.

The liver and spleen were large and congested, they contained no malarial pigmented bodies.

The mesenteric glands were not swollen.

The small intestine and colon were normal.

The kidneys showed the lesions of acute degeneration.

CASE LIV. (Figure 29.) ***Both Malarial and Typhoid Poisoning; Irregular Temperature; Death.***

Male, 25, had been sick in Cuba and came into the hospital at Montauk thirteen days before his death. The only symptoms were the prostration, the irregular fever seen in the chart, and a profuse diarrhœa.

Autopsy.—The brain was not examined.

The heart was normal.

The lower lobes of both lungs were congested with commencing red hepatization.

The liver and spleen were large and dark-red colored. In the blood from the spleen there was pigment and a few red cells contained rings.

The mesenteric glands were swollen. In the ileum, just above the cæcum, there were cicatrizing ulcers, farther up there were fresh ulcers, and still farther up swollen agminated glands.

THE MILD FORM.

The pulse and temperature remain nearly or quite normal. The patients have irregular feelings of heat and cold, headache, irritability, sleeplessness, loss of appetite, and general prostration and discomfort, but are not sick enough to be in bed. The bowels continue regular, or there may be constipation, or diarrhœa.

In some of the patients bronchitis is a prominent symptom, in others nausea and vomiting.

The patients remain in this condition for three or four weeks. They are liable to hemorrhage from the intestine, to perforation of the intestinal ulcers, and to attacks of delirium.

THE FATAL CASES.

Deaths from bleeding from intestinal ulcers and deaths from perforation we have to expect as yet as unavoidable. Deaths from broncho-pneumonia, lobar pneumonia, or colitis, are infrequent.

The most important deaths are those from uncomplicated typhoid fever. They are not nearly as numerous as they used to be, but they still occur. I think it is important to give several examples:

CASE LV. (Figure 30.) *Typhoid Fever, Uncomplicated; Death on the Twenty-second Day.*

The patient was a girl of 15, previously in good health, who went through her illness in a private house, with the best possible care and nursing, and systematic cold tubbing for the first two weeks. The disease was not controlled at any time, there were no complications; the complete chart tells the story.

CASE LVI. *Typhoid Fever Fatal on the Ninth Day, with an Extensive Intestinal Lesion.*

Male, 18 years old, nine days before death had a chill, followed by fever. The next day he had headache, stiffness of the muscles of the back of the neck, diarrhœa, and tympanites.

Five days before death the face was flushed, pupils contracted, delirium, twitching of the muscles, diarrhœa, the temperatures ran irregularly from 102° to 106°, the pulse from 122 to 130.

He died on the ninth day of the disease.

Autopsy.—The brain was not examined.

The heart was normal.

The lungs were congested.

The liver was normal.

The spleen was large and soft.

The kidneys were pale.

The mesenteric glands were much swollen.

In the ileum most of the solitary and agminated glands were converted into ulcers with thick edges and yellow, sloughy centres.

The upper part of the colon was congested.

CASE LVII. *Death on the Tenth Day Before Ulceration.*

Male, 21 years old, one week before admission began to have headache, vertigo, chills, fever, pain in the abdomen and diarrhœa.

Eighth day: A strong, well-nourished man, face flushed, tongue coated, mind dull, diarrhœa, tympanites, sibilant and sonorous râles over both lungs, temperature 103.4°, pulse 89.

Ninth day: Delirious, twitchings of muscles of face, involuntary passages. Death on the tenth day.

Autopsy.—The pia mater and the brain itself were congested.

The heart was normal.

The lungs were congested.

The liver was large and pale.

The spleen was large and firm.

Through the whole length of the ileum there was marked swelling without ulceration of the solitary and agminated glands.

The mesenteric gland was swollen.

The colon was normal.

The kidneys were large and pale.

CASE LVIII. *Death on the Twelfth Day; Moderate Constitutional Symptoms with Very Extensive Intestinal Lesions.*

Male, 22 years old, on August 1 began to have diarrhœa; some of the passages were fæcal, some were streaked with blood. He had no other symptoms and did not feel ill. On August 6, for the first time, he remained in bed, his pulse was 100, temperature 99°, he had pain in the abdomen and diarrhœa.

August 7: Pulse 100 to 110, temperature 100° to 101.5°, tongue dry, no diarrhœa.

August 10: Pulse 100 to 110, temperature 101° to 102°.

August 11: Pulse 110 to 125, temperature 101° to 102°, delirious, large passages of blood from the bowels.

August 12: He died.

Autopsy.—For nearly the whole length of the ileum the solitary and agminated glands were very much swollen and ulcerated.

In the colon for its entire length the solitary glands were swollen and ulcerated. In the stomach there were a number of small round ulcers.

CASE LIX. *Death on the Fourteenth Day; Extensive Intestinal Lesion.*

Female, 30 years old, eight days before admission had chills, fever, abdominal pain, vomiting, cough and mucous sputum.

Ninth day: Tongue brown and dry, coarse râles over both lungs, tympanites, delirium, temperature 102.5°.

She went on with temperatures between 101° and 104°, and died on the fourteenth day.

Autopsy.—The brain was normal.

The heart was normal.

There was muco-pus in the bronchi.

The liver was normal.

The kidneys were large and congested.

The spleen was normal.

The mesenteric glands were swollen.

In the ileum the solitary and agminated glands were converted into ulcers, with thick edges and adherent yellow sloughs.

CASE LX. *Death on the Fourteenth Day; Fairly Marked Intestinal Lesion.*

Male, 16 years old, on July 27 had a severe chill, fever, headache, cough and mucous sputum.

July 28: Diarrhœa.

July 29: Two chills.

August 4: Tongue dry and coated, diarrhœa, temperature 104°.

The temperature continued between 102° and 105°; on August 9 he died.

Autopsy.—The pia mater of the brain was a little congested.

The heart was normal.

The lungs were congested.

The liver was normal.

The spleen was large and soft.

The cortex of the kidneys was thick and white.

In the lower end of the ileum the solitary and agminated glands were much swollen and partly eroded.

The mesenteric glands were swollen.

CASE LXI. *Death on the Sixteenth Day with a Small Intestinal Lesion; Treated with Cold Tubs from Eighth to Fifteenth Day.*

Male, 30 years old, fifteen days before death had a severe chill followed by fever.

Eighth day: Face flushed, diarrhœa, very weak, temperature 105°, urine 1030, no albumin or casts. He was given tubs at 76° for half an hour each; the tubs reduced the temperature for from two to three degrees, but the patient was no better for them.

Ninth day: The eruption on the abdomen was well marked.

Eleventh day: Delirious, œdema of the lungs, for which he was dry-cupped. He died on the sixteenth day with great dyspnœa.

Autopsy.—The brain was not examined.

The heart was normal.

The lungs were congested and œdematous.

The liver was pale and soft.

The spleen was large and soft.

The mesenteric glands were but little swollen.

The kidneys showed the changes of acute degeneration.

In the lower end of the ileum, in only one agminated gland.

there was a large, deep ulcer beginning to cicatrize. The other agminated and the solitary glands were not affected.

The colon was normal.

CASE LXII. (Figure 31.) *Death on Sixteenth Day; Small Intestinal Lesion; Thrombus in Right Ventricle; Treated with Cold Tubs.*

Male, 24 years old, sixteen days before death was seized, after dinner, with an attack of vertigo and partial unconsciousness. After recovering from this he began to have a headache, which has continued ever since. He has had one or two attacks every day of vertigo, sometimes accompanied by loss of consciousness.

Fifth day: The temperature chart begins.

Sixth day: He is put on the regular treatment with cold tubs.

Ninth day: The eruption appeared on the abdomen, no diarrhœa.

Twelfth day: Began to be delirious.

Sixteenth day: He died.

Autopsy.—There was an increase of serum in the pia mater of the brain. In the right ventricle of the heart there was a firm, white, ante-mortum clot about the size of a chestnut.

The bronchi were moderately congested.

The stomach and liver were normal.

The spleen was large and soft and contained three infarctions.

In the ileum, eighteen inches above the ileo-cæcal valve, there was a swollen and congested agminated gland, above this three other agminated glands were swollen, none of them ulcerated.

The mesenteric glands were somewhat swollen.

The colon was normal.

The deaths which occur after typhoid fever, with clean, cicatrizing ulcers and no complications, are hard to account for.

CASE LXIII. (Figure 32.) *Death on the Forty-ninth Day with Cicatrizing Ulcers.*

Male, 12 years old, about three weeks before admission he had pain in the abdomen and cough with muco-purulent sputum. One week later he began to have headache and diarrhœa.

He entered the hospital on what was called the twenty-second day of his typhoid; the temperature chart begins on that day. He was given cold tubs from the twenty-fourth to the thirtieth day.

From the twenty-ninth to the thirty-fifth day he was better, then worse again.

Forty-sixth day: He passed blood from the bowels. After this there was almost constant bleeding from the bowels.

On the forty-ninth day he died.

Autopsy.—The heart was pale and flabby.

The lungs were œdematous.

The stomach was coated with mucus.

The liver was normal.

The kidneys were large, the cortex thick and pale.

The mesenteric glands were much swollen.

In the lower part of the ileum the agminated glands were changed into small, clean, cicatrizing ulcers.

Convalescence begins regularly in the fifth week. If uncomplicated it progresses steadily, but yet a number of months elapse before the patient is fit for much bodily or mental exertion.

The convalescence may be interrupted in several ways:

There may be severe pain in the back, apparently due to inflammation of the periosteum of the vertebræ.

The mind may remain dull, apathetic, and feeble for days or weeks.

There may be hemiplegia, dependent on thrombosis of the sinuses of the dura mater; or paralysis of groups of muscles due to changes in the peripheral nerves.

There may be hemorrhage from intestinal ulcers which have not healed, or perforation with peritonitis.

There may be a catarrhal gastritis with pain, nausea, and vomiting.

There may be catarrhal or croupous inflammation of the colon, with the symptoms of dysentery.

There may be rapid and irregular heart action.

The patients, after beginning to convalesce, may make no real progress and die in an emaciated and feeble condition.

POST-TYPHOID SEPSIS.

This is the most important disturbance of convalescence. Of the fevers which come after typhoid fever we distinguish:

1. The ordinary moderate rises of temperature lasting for a few hours occurring within three weeks after the end of a typhoid

fever. These short fevers would not be recognized at all unless the temperature was systematically taken, for they are not attended with any symptoms and do no harm. The only important thing about them is to understand that they are not of importance and must not be allowed to interfere with the regular management of the convalescence.

2. The post-typhoid fevers which are of real importance and last for one or more weeks, but yet do not make the patient very ill and are not fatal.

If one looks for this form of fever it is found to occur quite frequently. I have seen it after typhoid fevers of two, three, or four weeks' duration. The rise of temperature is continuous with that of the typhoid, or there is an interval of several days of normal temperature and then the post-typhoid fever begins.

The morning temperatures run pretty near the normal, the afternoon temperatures run from 100° to 103°, with occasional jumps to 105° or 107°. With the very high temperatures there may be chills.

With this fever there is no delirium or sleeplessness; the tongue is moist and clean; there is no eruption, no tympanites, no diarrhœa. The patients are hungry. If they are not properly fed they may have rather alarming attacks of heart failure. As long as the patients are kept in bed and on a fluid diet there is no particular limit to the duration of the fever, but yet in the end they get well.

CASE LXIV. (Figure 33.) *Three Weeks' Typhoid Fever; Sixteen Days' Mild Post-Typhoid Fever; Chart Begins on the Fourteenth Day.*

Female, 5 years old, fourteen days ago began to have headache, sore throat, diarrhœa and fever.

Fourteenth day: Apathetic, prostrated, tongue coated and moist, tympanites, urine 1012, trace of albumin. She was given cold tubs from the fourteenth to the seventeenth day.

Hæmoglobin 80 per cent., red cells 4,496,000, white cells 7,800.

Sixteenth day: Positive Widal reaction.

Twenty-third day: She was hungry and was given solid food.

Twenty-seventh day: She sat up in bed.

Thirty-third day: She was out of bed.

Fiftieth day: Hæmoglobin 86 per cent.

Fifty-third day: Discharged in very good condition.

CASE LXV. (Figure 34.) *Four Weeks' Typhoid Fever, Intermission of Seven Days, then Post-Typhoid Fever Until the Fifty-third Day; Treatment by Cold Tubs from the Eighteenth to the Twenty-third Day.*

Female, 13 years old, seventeen days before admission had a chill followed by fever, headache, vomiting and diarrhœa. The chart begins on the eighteenth day.

On the twenty-sixth day she was improving rapidly.

On the twenty-eighth day she began to have scraped beef.

On the thirty-fourth day she was out of bed.

On the thirty-eighth day the hæmoglobin was 88 per cent., the red cells 4,846,000, white cells 8,400.

On the forty-fifth day, although the temperature rose to 105°, she was not at all sick.

On the fifty-second day the hæmoglobin was 94 per cent., red cells 5,280,000.

The next day she was discharged.

CASE LXVI. (Figure 35.) *Three Weeks' Typhoid Fever; No Intermission; Post-Typhoid Fever Until the Forty-third Day. The Chart Begins on the Eighth Day.*

Male, 25 years old, ten days before admission began to have headache, and went to bed four days ago. He was treated with cold tubs from the eighth to the fourteenth day.

He was fed on the twenty-second day, taken out of bed on the thirtieth day and discharged on the forty-ninth day.

By the thirty-fourth day he was feeling well and rapidly gaining flesh and strength. On the forty-first day the hæmoglobin was 70 per cent., red cells 4,722,000.

CASE LXVII. (Figure 36.) This was a two weeks' typhoid fever with a positive Widal reaction. There was an intermission of six days. Then on the twenty-first day the temperature ran up to 105°, on the twenty-second and twenty-third days to 107°. With these rises of temperature there were chills and sweating with some prostration. There was no thrombosis of the veins. On the twenty-fourth day he was doing well, on the twenty-eighth day he was out of bed, and on the forty-sixth day he was discharged.

It is very interesting in these patients, if they are fed and taken out of bed, to see how they steadily gain flesh and strength in spite of the fever.

3. Much more important than these mild fevers are the severe

and long-continued post-typhoid fevers, which may terminate fatally. They occur regularly after well-marked and severe typhoid fevers, which run their full four weeks' course. The septic fever is continuous with the typhoid fever. Sometimes it even overlaps it, so that both fevers seem to be going on together in the fourth or fifth week.

The morning temperatures run about 100° and the afternoon temperatures about 103°. In many cases there are sudden jumps of temperature up to 105°, 106°, or 107°. These jumps occur in the course of the regular fever, or there are intervals of one or more days with nearly normal temperatures and then the sudden rise. With these sudden rises of temperature there are often chills.

It is noticeable that the septic temperature is not controlled by baths, as in typhoid temperature, but runs up and down quite without reference to them.

The patients look septic and not in the typhoid state. The mind is often dull and apathetic, sometimes with delusions, but not often with active delirium. The tongue is dry, there may be nausea and vomiting, and yet there is some desire for food. There is a rapid loss of flesh and strength as in ordinary sepsis. In some of the cases there is thrombosis of one or both femoral veins. If the patients are insufficiently fed and kept in bed they are very apt to die.

In the autopsies of the fatal cases, which I have made, the intestinal lesions were healed and there seemed to be no cause of death but sepsis and starvation.

Some of the patients improve on food alone, but in most of them there is no real convalescence until they are taken out of bed. Then they gain flesh and strength even while the fever continues.

It must be confessed that it requires a good deal of courage to take out of bed a patient who is apparently dying; but I am fully convinced that the only way of saving the lives of the bad cases is to feed them and take them out of bed.

CASE LXVIII. (Figure 37.) *Severe Post-Typhoid Sepsis.*

Female, 26 years old, fifteen days ago began to have headache, nausea, diarrhœa and fever.

Sixteenth day: The chart begins. She was prostrated, tongue red and dry, râles over both lungs, moderate tympanites, urine 1020, trace of albumin, a few granular casts, put on tub baths.

Seventeenth day: Chill, vomiting, positive Widal reaction.

Eighteenth day: She looks very sick, delirious, diarrhœa.

Twentieth day: Diarrhœa, the tub baths at 60° are badly borne.

Twenty-eighth day: She continues to be very ill, a little blood in the stools, the tub baths are stopped.

Thirty-first day: She looks septic.

Forty-third day: She continues to be very ill, nausea, vomiting and diarrhœa.

Forty-sixth day: She was given meat and enjoyed it. After this she steadily improved.

Fifty-third day: Hæmoglobin 60 per cent., red cells 3,586,000, white cells 6,600.

Fifty-fourth day: Out of bed.

Sixty-eighth day: Hæmoglobin 65 per cent., red cells 4,080,000. Between the fifty-eighth and the sixty-fifth days she gained fifteen pounds in weight.

CASE LXIX. (Figure 38.) *Severe Post-Typhoid Sepsis after Four Weeks' Typhoid.*

Male, 31 years old, two weeks ago began to have chill, fever, headache and diarrhœa; for ten days he has been in bed. The chart begins on the fifteenth day. He was treated with cold tubs from the fifteenth to the thirty-second day.

Twenty-third day: Positive Widal reaction.

Twenty-sixth day: He is sleeping well and is hungry in spite of the temperature. He continued to improve until the fortieth day, when he was given solid food.

Forty-sixth day: He was out of bed.

Fiftieth day: He weighs 102 pounds.

Sixty-fifth day: Hæmoglobin 58 per cent., red cells 4,444,000, white cells 4,900.

Seventy-second day: Hæmoglobin 57 per cent., red cells 3,608,000, white cells 5,800.

Seventy-eighth day: He weighs 123 pounds.

Eighty-sixth day: Hæmoglobin 77 per cent., red cells 5,328,000.

CASE LXX. (Figure 39.) *Severe Post-Typhoid Sepsis Overlapping Typhoid Fever; Thrombosis of the Femoral Veins.*

Male, 29 years old, five days before admission had chill, headache, vomiting and fever.

The chart begins on the sixth day; there was a positive Widal reaction on the eighth day. He was treated with cold tubs from the sixth to the forty-third day.

His typhoid fever was severe until the eighteenth day when he was evidently less poisoned. On the twenty-fifth day he had a chill, the temperature ran up to 107° and he looked septic. There was no thrombosis of the veins, the white blood cells were 6,900. Apparently the septic poison was overlapping the typhoid. He also had nausea and vomiting. He went on after this with irregular afternoon temperatures, often up to 104°, not at all controlled by tubbing, until the forty-third day, when the tubs were stopped. They had been of absolutely no use after the twenty-first day.

On the forty-fifth day he was fed.

On the forty-seventh day, although he was so ill and weak it seemed doubtful if he would recover, he was taken out of bed. After this the temperatures were very irregular, running up to 106° and down to normal. There was no real improvement until the fifty-first day. After this he improved steadily, although there was thrombosis of the left femoral vein on the fifty-ninth day and of the right femoral vein on the sixty-first day. There was no more fever after the sixty-eighth day. He gained thirty pounds in weight between the sixty-ninth and the one hundred and fifth day.

CASE LXXI. (Figure 40.) *Severe Post-Typhoid Sepsis; Thrombosis of the Femoral Vein; Orchitis.*

Male, 32 years old, entered the hospital on the tenth day of his typhoid; the chart begins on that day. He was treated with cold tubs from the tenth to the nineteenth day, with three-grain doses of acetanilid from the twentieth to the thirty-third day.

He ran a pretty severe typhoid, but began to improve on the twenty-fifth, and was given scraped beef on the twenty-sixth day. He continued to improve, although the afternoon temperatures were up to 104°, until the thirty-third day. Then he had a severe chill, the temperature ran up to 106°, there was thrombosis of the left femoral vein and he looked distinctly septic. After this he failed steadily, lost flesh and strength, and seemed likely to die.

On the forty-third day, with much misgiving, for it seemed like handling a dying man, I got him out of bed. The chart shows that during the following week the temperature did not go over 101° and his general improvement was most satisfactory. After this he got steadily better, taking more and more solid food, although he had irregular rises of temperature and a complicating orchitis. He was discharged well on the eighty-third day.

Case LXXII. (Figure 41.) *Post-Typhoid Sepsis; Repeated Chills; Thrombosis of the Femoral Veins.*

Male, 17 years old, four days before admission had headache, fever, prostration, and at once went to bed.

On the fifth day the chart begins. He was put on cold tubs from the sixth to the eleventh day.

From the fourteenth to the twenty-fourth day he was doing well and getting hungry, although the temperature remained high.

Twenty-fifth day: Thrombosis of the left femoral vein.

Twenty-seventh day: Chill, followed by rise of temperature to 105°.

Twenty-eighth day: Two chills, temperature up to 106°.

Twenty-ninth day: Two chills, temperature up to 106°.

Thirtieth day: One chill, temperature 103°.

Thirty-eighth day: Thrombosis of right femoral vein.

Thirty-ninth day: He was given scraped beef.

Forty-first day: He is improving, hæmoglobin 85 per cent., red cells 4,540,000, white cells 21,600.

Forty-third day: He is out of bed.

Fifty-fourth day: Hæmoglobin 65 per cent., red cells 3,976,000, white cells 11,000.

Seventy-eighth day: Discharged in good condition.

Case LXXIII. (Figure 42.) *Post-Typhoid Sepsis After a True Relapse.*

This man had a typhoid fever lasting for four weeks, then there was an intermission of three days. Then there was a true relapse lasting for ten days. During the relapse he began to become septic and developed thrombosis of the right femoral vein on the thirty-eighth day. In the seventh, eighth and ninth week the rises of temperature were curious, with intervals of one, two, three, and four days between them. There were no malarial organisms in the blood. On the fifty-seventh day the hæmoglobin was 70 per cent., red cells 4,608,000, white cells 4,400. He was fed on the thirty-seventh day, taken out of bed on the fifty-second day, and discharged on the seventy-fourth day. He gained ten pounds in weight between the fifty-second and the sixty-ninth day.

Case LXXIV. (Figure 43.) *Severe Post-Typhoid Sepsis; Thrombosis of External Iliac Vein and of Sinuses of Dura Mater.*

Male, 50 years old, began his typhoid with an acute invasion and went on with the ordinary symptoms of a severe typhoid. The chart began on the seventh day; I saw him for the first time on the twentieth day. He was in his own apartment and was very well nursed, but received no medical treatment or baths.

Twentieth day: He looked very ill indeed with a sullen, suspicious delirium. On that day he had two chills followed by sweating.

From the twenty-second to the twenty-seventh days there was the falling temperature of the fourth week of typhoid, but there was no improvement in his general condition and the delirium continued.

Twenty-eighth day: The temperature suddenly rose to 104.5°.

Twenty-ninth day: There was a chill lasting for half an hour, followed by thrombosis of the left external iliac, with a great deal of swelling of the whole leg and lower part of the back.

Thirty-fifth and thirty-sixth day: Marked chills.

Thirty-seventh to the thirty-ninth day: He looked as if he would not recover. He was unconscious, with general muscular contraction. I believed there was thrombosis of some of the smaller veins of the dura mater.

Fortieth day: He had a chill followed by sweating.

Forty-first day: I gave him a poached egg, and after this gradually got in more and more solid food.

Forty-second day: A chill.

Forty-fourth day: A chill.

Forty-fifth day: The temperature fell to 97° for a short time. I began to treat the swollen leg with systematic rubbing and bandaging.

Forty-sixth day: A chill, the temperature fell to 97°. He sat up in a chair for twenty minutes; his mind was now clear. After this he was up in a chair every day.

On the forty-ninth and fifty-first days the temperatures ran from 105° to 97°.

He had been making slow but real improvement, and on the fifty-third day he was helped to walk into the next room.

Fifty-eighth day: He had his chill and high temperature for the last time.

After the sixtieth day he had no more fever.

Sixty-fifth day: He was moved from his apartment to a hotel.

After this convalescence was uninterrupted, the improvement in the swollen leg being unusually rapid.

Treatment.—The principles of the prevention of typhoid fever seem to be well established, but the continued frequency of the disease in so many different countries would seem to show that there are still some gaps in our knowledge.

The fæces, the urine, and the soiled clothes of typhoid fever patients all contain typhoid bacilli and must be disinfected or destroyed.

The patient himself, under ordinary conditions, is not dangerous. But in camps and armies experience shows that they should be isolated.

It has been demonstrated that typhoid bacilli are introduced into the body with contaminated water, ice, milk, and food, and cause the disease. These sources of disease can in large measure be guarded against. The reduction in the number of typhoid cases in cities by purifying the water supply has been very marked.

A considerable amount of work has been done with preventive inoculations of attenuated cultures of the typhoid bacillus in the British army in India and South Africa. The results are still uncertain.

It would seem that after the long number of years that typhoid fever has prevailed in many different countries that there might be some general agreement as to its management and treatment. This, however, is not the case; many different ideas still prevail. These can be grouped as follows:

1. Specific treatment with serums. This has not advanced beyond the experimental stage.

2. Expectant treatment, with treatment directed to the relief of symptoms. This method has been and still is employed in a large number of cases. The results can be very satisfactory, but depend largely on the intelligence and experience of the physician in charge.

3. Treatment directed to the condition of the small intestine. This method is based on the idea that the typhoid bacilli live, grow, and evolve their toxin in the cavity and wall of the lower part of the small intestine.

Originally antiseptic drugs were given with the idea that they were competent to exert a direct bactericidal effect on the specific organisms present in the intestine.

Now the matter is stated somewhat differently. "It is said that antiseptics are not given in the belief that they are competent either to slay the germ of typhoid fever or to neutralize its toxin, but in the confident anticipation that they will exert a restraining influence on the propagation of the specific bacillus and on the various putrifactive organisms which are associated with it in the alimentary canal. Moreover, even if antiseptic remedies are incompetent to exercise any inhibitory influence on the multiplication of bacilli which have already passed into the circulation and have been conveyed to distant organs, we are prepared to believe that some degree of attenuation of these organisms may be brought about by the presence of even minute quantities of an antiseptic in the blood and tissues of the body.

"To obtain the full benefit of the antiseptic method it is necessary that the remedies should be given early in the disease and in sufficient doses. Under favorable conditions it is claimed that the duration of the disease will be shortened and the intensity of the fever lessened; that the mouth and tongue will be more clean and moist; that diarrhœa will be controlled, tympanites held in check, bleeding and perforation be less frequent and the duration of convalescence shortened."

All of this is believed by many men of intelligence and large experience. If it were true it would be very satisfactory.

It is hardly necessary to enumerate the antiseptics that have been employed; they range from calomel to the oil of cinnamon.

4. Treatment directed to the lowering of the temperature. It has been believed by many that by far the greater number of those who succumb to typhoid fever die from the effect, directly or indirectly, of the fever heat. It has followed, therefore, that antipyretic treatment has been very extensively used.

An antipyretic effect may be produced either by drugs or by the direct application of cold to the surface of the body.

The drugs most frequently given are: quinine, antipyrine, acetanilid, phenacetine, and a number of others of the same class.

Cold is applied to the surface of the body by sponging, sprinkling, wet packs, cold tubs and ice.

In practice it is easy to reduce the temperature in these different ways; it is not so easy to exert any real effect on the disease.

5. Treatment of typhoid fever as a disease with cold tubs. This method, if carried on exactly according to the instructions

of Brand, immersion of the whole body in water at 65° every three hours if the temperature reaches 103°, with friction, is unquestionably a real treatment of the disease, and has diminished its mortality very markedly.

It has its disadvantages. It is difficult and expensive to carry out, it is very disagreeable to the patient, there are patients in whom the reduction of temperature is excessive, and the treatment does harm instead of good.

CASE LXXV. (Figure 44.) *Three Weeks' Typhoid; Treatment by Cold Tubs; Excessive Falls of Temperature; Recovery.*

Male, 21 years old, eight days ago lost appetite, was sleepless, but was not aware of having fever.

Three days ago he vomited, had abdominal pain, bowels constipated, great prostration.

Fourth day: Face flushed, tongue coated and dry, no tympanites, no eruption, urine 1018, no albumin or casts. Ordered tub baths at 60° F. every three hours if the temperature reaches 103° F.

Fifth day: Shivering and cyanosis after each bath.

Ninth day: Typical eruption over abdomen, diarrhœa.

Thirteenth day: Looks better, tongue is clean, is hungry.

Eighteenth day: Baths were stopped, was improving, was given scraped beef.

Twenty-second day: Sleeps well, sits up in bed, has chops and potatoes.

Twenty-third day: Out of bed.

Twenty-eighth day: Left the hospital.

6. The general management of the patients. It is agreed on all hands that the patients should be kept in bed and on a fluid diet. Some preparation of milk is the food ordinarily used.

There are, however, physicians who give their patients nothing but water during the first two weeks of the disease; and it must be admitted that such a starvation plan is often attended with low temperatures and sometimes with recovery.

The main points of divergence are as to the use of alcoholic stimulants, and the proper time for giving solid food.

As to the use of alcoholic stimulants different views prevail: some give them as a routine throughout the disease; some only give them when the heart's action is feeble; some think that as a rule they do harm rather than good.

In the same way as regards the time for giving solid food. Many believe that no solid food should be given until the temperature has been normal for two weeks; that if this rule is disregarded the dangers of relapse, perforation and bleeding are increased.

Others, on the contrary, think that as soon as the tongue is moist and clean and there is hunger, that meat in small quantities should be given; that if this rule is disregarded the risk of heart failure is increased and convalescence is deferred.

PERSONAL OBSERVATIONS.

I have observed typhoid fever in New York since 1866. I have had the opportunity of seeing the course of the disease in cases under my own care and under the care of other physicians. I have made many autopsies. I have naturally formed some definite ideas about the disease:

1. The disease is not nearly as fatal in New York as it formerly was. The following table shows the change in the hospital mortality:

TABLE OF THE MORTALITY FROM TYPHOID FEVER IN NEW YORK HOSPITALS DURING THE YEARS 1877 TO 1883, AND THE YEARS 1900 TO 1904.

Hospitals.	1877 to 1883.			1900 to 1904.		
	Cases.	Deaths.	Mortality.	Cases.	Deaths.	Mortality.
Bellevue.............	311	101	32.4%	600	93	15.5%
Mt. Sinai............	169	29	17.1%	425	36	8.4%
New York.	107	23	21.4%	571	53	9.2%
Roosevelt............	147	44	29.9%	584	76	13.0%
St. Luke's...........	85	22	25.8%	486	64	13.6%
Total............	819	219	26.7%	2,648	322	12.1%

I should say the mo[illegible] in priva[illegible]tice is not over 5 per cent.

2. In the fatal cases death is now regularly due to bleeding or perforation—not often to typhoid fever as a disease.

3. This change in the mortality is not due to a change in the type of the disease, but to improvement in care and nursing and the use of tubbing.

4. A satisfactory plan of treatment is one which not only saves the life of the patient but also alleviates the course of the disease and shortens convalescence.

5. Calomel does no good and not infrequently causes salivation.

Alcoholic stimulants usually do harm; sometimes they are necessary.

The antipyretic drugs may be of use in the third and fourth weeks of the disease, not earlier.

The application of cold to the surface of the body by sponging, sprinkling, slush baths, etc., has no effect on the course of the disease.

While the disease is going on the best food for the patient is milk. As soon as the disease is through with they should have scraped beef. As convalescence progresses the quantity of food must be increased.

6. I have seen many patients do very well with the expectant and antiseptic plans of treatment.

There can be no question that the mortality of the disease has been much decreased since the use of cold tubbing has become general. I think we are learning that the good effects are produced by the immersion of the body in a large quantity of water, and that the water need not be cold.

7. Personally, I like to begin in the first week with tubs of 99° F. at 12 M., 4 P.M., and 8 P.M., whether the temperature is high or low. These tubs I continue to the end of the second week or a little longer, and then stop. In the fourth week the natural breaks in the temperature may be made greater by the use of small doses of acetanilid.

We cannot expect the patients to take much milk during the first week, but after that an adult should take eight ounces every three hours. If the heart becomes feeble during the fourth week scraped beef is a better stimulant than whiskey or digitalis.

Sulfonal, trional and veronal seem to be the best hypnotics.

The objection to opium is that it is apt to cause retention of urine.

Excessive tympanites is best treated by rectal irrigations with hot water.

For intestinal bleeding the ice coil over the abdomen is indicated.

For perforation of the intestine the only hope is in an early operation.

In cases of post-typhoid sepsis it is of the greatest importance that the patients should be given solid food and taken out of bed.

List of Cases and Charts.

Case I. (Figure 1.) Complete chart; gradual invasion; four weeks' typhoid; treatment by intestinal antiseptics; mild post-typhoid sepsis.

Case II. (Figure 2.) Complete chart; acute invasion; two weeks' typhoid; no treatment; recovery.

Case III. (Figure 3.) Complete chart; acute invasion; four weeks' typhoid; treatment with warm tubs.

Case IV. (Figure 4.) Complete chart; acute invasion; four weeks' typhoid; cystitis; treatment with warm tubs.

Case V. (Figure 5.) Invasion with repeated chills; low and irregular temperature; rapid pulse; death on the nineteenth day; small intestinal lesion.

Case VI. (Figure 6.) Invasion with bronchitis; three weeks' typhoid: irregular temperature; recovery.

Case VII. (Figure 7.) Complete chart; invasion with pleurisy with effusion; four weeks' typhoid; the patient kept sick with whiskey and bad treatment.

Case VIII. (Figure 8.) Invasion with vomiting and diarrhœa.

Case IX. (Figure 9.) Complete chart; invasion with acute exudative nephritis; treatment by Woodbridge method; three weeks' typhoid; recovery from the typhoid and the nephritis.

Case X. (Figure 10.) Invasion like that of cerebro-spinal meningitis; excessive falls of temperature from cold tubs; death on the seventeenth day; deep ulcers in the ileum; peritonitis without perforation.

Case XI. Typhoid fever with cerebral symptoms.

Case XII. (Figure 11.) Complete chart. Mild typhoid for two weeks; temporary improvement; more severe typhoid for three weeks; recovery.

Case XIII. (Figure 12.) Irregular invasion; irregular course; purulent bronchitis; death.

Case XIV. Death from syncope.

Case XV. Early and excessive tympanites; recovery.

Case XVI. (Figure 13.) Five weeks' typhoid; repeated intestinal bleedings; recovery.

Case XVII. (Figure 14.) Four weeks' typhoid; repeated bleedings; recovery.

Case XVIII. Fatal bleeding from the bowels on the seventh day.

Case XIX. (Figure 15.) Four weeks' typhoid; bleeding from the bowels on the twenty-third day; convalescence on the twenty-ninth day; fatal bleeding on the thirty-seventh day.

Case XX. (Figure 16.) Death on the thirteenth day from bleeding from the ileum into the colon, the blood not passed during life.

Case XXI. Fatal bleeding on the thirteenth day.

Case XXII. Staphylococcus sore mouth on the sixteenth day of a four weeks' typhoid.

Case XXIII. Broncho-pneumonia with typhoid fever; death on the seventh day.

Case XXIV. Typhoid fever; lobar pneumonia; death.

Case XXV. Typhoid fever; hemiplegia; recovery.

Case XXVI. Perforation; peritonitis; death.

Case XXVII. Perforation during convalescence; death.

Case XXVIII. Perforation of the appendix after typhoid fever.

Case XXIX. Peritonitis during convalescence due to an inflamed mesenteric gland.

Case XXX. Acute colitis after typhoid fever; recovery.

Case XXXI. Acute colitis after typhoid fever; recovery.

Case XXXII. (Figure 17.) Croupous colitis after typhoid; death.

Case XXXIII. Amœbic colitis after typhoid fever; death.

Case XXXIV. Typhoid fever with jaundice.

Case XXXV. Epididymitis and orchitis on the nineteenth day of typhoid fever.

Case XXXVI. Periostitis on the twelfth day of typhoid fever.

Case XXXVII. Periostitis during convalescence.

Case XXXVIII. Arthritis of the wrist joint during convalescence.

Case XXXIX. (Figure 18.) Two weeks' typhoid fever; no treatment.

Case XL. (Figure 19.) Two weeks typhoid; irregular temperature; bleeding from the bowels; no treatment.

Case XLI. (Figure 20.) Two weeks' typhoid; Woodbridge treatment; recovery.

Case XLII. (Figure 21.) Six weeks' typhoid; chart from the fourth to the forty-third day; no treatment; recovery.

Case XLIII. (Figure 22.) Eleven weeks' typhoid; treatment by cold tubs; period of improvement in the sixth week; recovery.

Case XLIV. (Figure 23.) Typhoid fever with low morning temperatures; cold tubs for two days.

Case XLV. (Figure 24.) Four weeks' typhoid; temporary fall of temperature while the disease was going on.

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Case XLVIII. (Figure 27.) Double relapse.

Case XLIX. Walking typhoid fever; death.

Case L. Walking typhoid fever; death.

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Case LII. Malarial fever followed by typhoid fever.

Case LIII. Malarial fever, or typhoid fever; death; no lesions.

Case LIV. (Figure 29.) Both malarial and typhoid poisoning; irregular temperature; death.

Case LV. (Figure 30.) Complete chart; uncomplicated typhoid fever; treatment with cold tubs; death on the twenty-second day.

Case LVI. Typhoid fever fatal on the ninth day; extensive intestinal lesion.

Case LVII. Typhoid fever fatal on the tenth day, before ulceration.

Case LVIII. Death on the twelfth day; moderate constitutional symptoms with extensive intestinal lesions.

Case LIX. Death on the fourteenth day; extensive intestinal lesions.

Case LX. Death on the fourteenth day; fairly marked intestinal lesion.

Case LXI. Death on the sixteenth day with a small intestinal lesion; treatment with cold tubs.

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Case LXIV. (Figure 33.) Mild post-typhoid sepsis.

Case LXV. (Figure 34.) Mild post-typhoid sepsis.

Case LXVI. (Figure 35.) Mild post-typhoid sepsis.

Case LXVII. (Figure 36.) Mild post-typhoid sepsis.

Case LXVIII. (Figure 37.) Severe post-typhoid sepsis.

Case LXIX. (Figure 38.) Severe post-typhoid sepsis.

Case LXX. (Figure 39.) Severe post-typhoid sepsis overlapping typhoid fever; thrombosis of the femoral veins.

Case LXXI. (Figure 40.) Severe post-typhoid sepsis; orchitis; thrombosis of the femoral veins.

Case LXXII. (Figure 41.) Severe post-typhoid sepsis; repeated chills; thrombosis of femoral veins.

Case LXXIII. (Figure 42.) Post-typhoid sepsis after a relapse.

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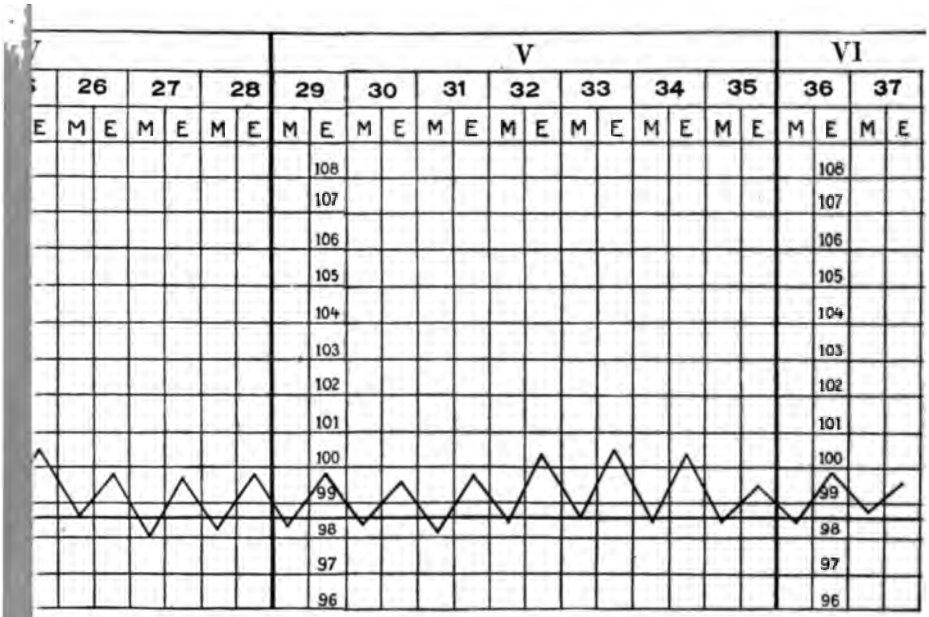
Case LXXV. (Figure 44.) Excessive effect of cold tubs in reducing temperature.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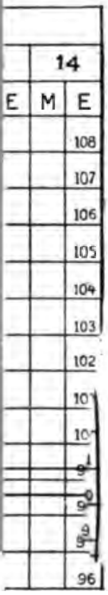
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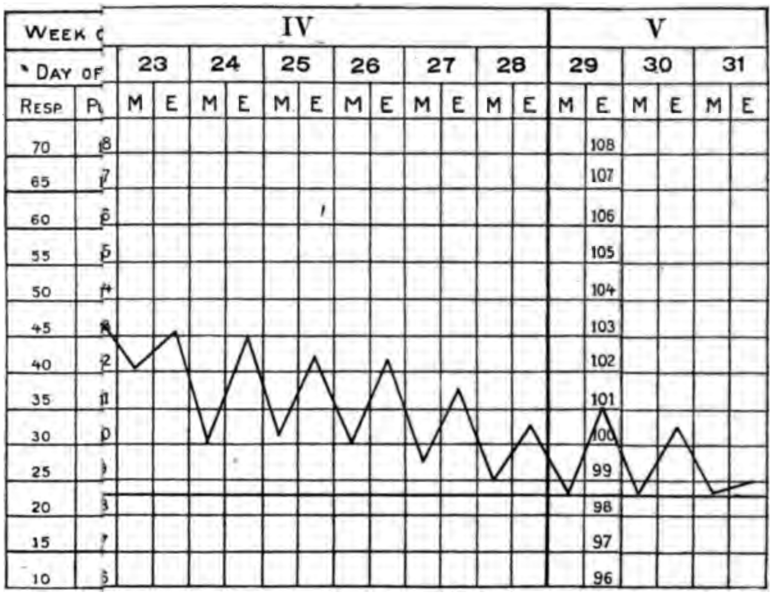
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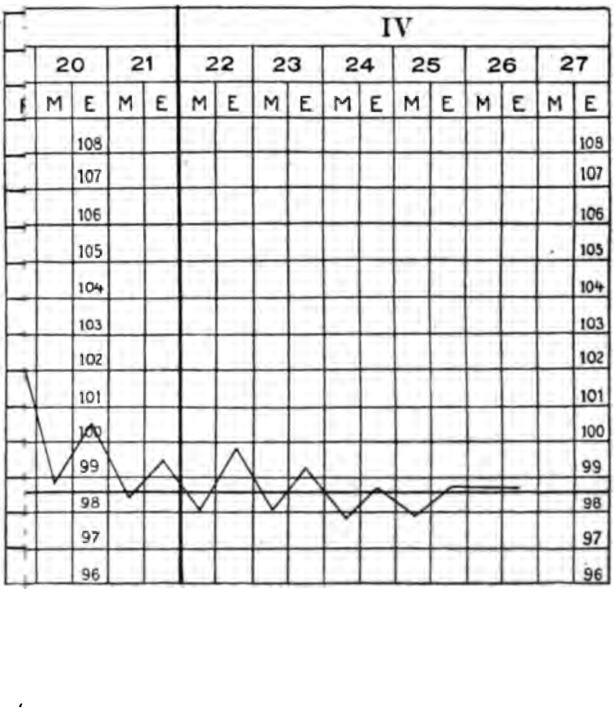
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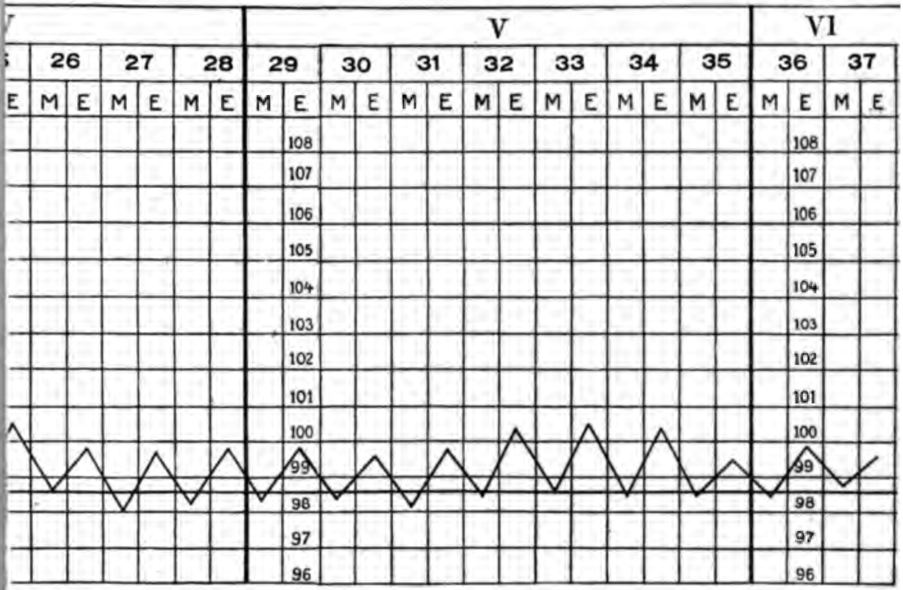
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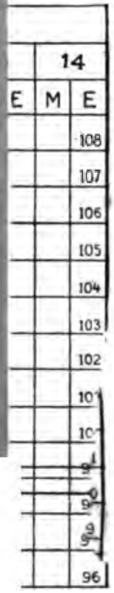
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LECTURES

ON

THE PRACTICE OF MEDICINE

WITH CASES AND CHARTS

BY

FRANCIS DELAFIELD, M.D., LL D. (Yale)

PART VI

INFLUENZA 607 RHEUMATISM 623
IRREGULAR FEVERS 636 THE LARGE INTESTINE 654
THE STOMACH 716 THE LIVER 773

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EPIDEMIC INFLUENZA.

Synonyms.—Epidemic catarrhal fever; grippe.

Definitions.—An infectious disease, caused by the presence and growth of the influenza bacillus, characterized by a rise of temperature, prostration, pains and inflammations of different parts of the body. The parts of the body which may be inflamed are: the pia mater, the peripheral nerves, the mucous membranes of the nose, throat, larynx, bronchi, stomach, and colon, the lungs, the pleura, the pericardium, the endocardium, the muscles and the skin.

History and Causes (Ziemssen).—The history of influenza can be traced back with certainty only to the beginning of the sixteenth century. With the year 1510 begins a series of epidemics the wide distribution of which has been reached by no other acute infectious disease. Up to the year 1870 more than ninety epidemics have been described, which generally extended over whole countries, or whole quarters of the earth. As a rule, the epidemics advance from the east and northeast toward the west. Sometimes they occur simultaneously at several different parts of the earth. The epidemics lasted, as a rule, only four to six weeks; only exceptionally, as in Paris, in 1831, have they lasted for nine or ten months. Wherever the epidemics prevail, very large numbers of the population are attacked There is no special liability or immunity by reason of age, sex, constitution, or condition of life; but in some epidemics children are exempt. The influenza, as such, is not usually fatal, but while it prevails the general death rate is increased. Climate and atmospheric influences seem to exert no effect. One attack of the disease does not protect against subsequent attacks.

In New York since 1890 the disease has been fairly established. In the summer there are few cases, but in the fall, winter and spring it is very prevalent. The years have varied as to the number of cases, the type of the disease and its severity.

Lesions.—The bacillus which is believed to be characteristic of influenza has been found in the sputum, the lungs, and the blood. The inflammations which it causes are of exudative type.

The changes in the mucous membrane of the conjunctiva, nose, throat and larynx are the congestion, swelling and increased production of mucus that belong to ordinary acute catarrhal inflammations.

The bronchitis is regularly extensive and severe with a large production of mucus, pus cells and sometimes blood. It is to be noted that in New York in nearly every case of acute or chronic bronchitis influenza bacilli can be found in the sputum, although no other symptoms of influenza are present.

The pleurisy and the pericarditis may be with fibrin alone, with fibrin and serum, or with fibrin, serum and pus.

In New York the pneumonias that occur with influenza are regularly due to a double infection with influenza bacilli and pneumococci. The influenza bacillus seems to belong to the bronchitis and the pneumococcus to the inflammation of the lung. In children the anatomical type is that of broncho-pneumonia. In adults the pneumonia involves one or more lobes with complete or incomplete hepatization, often with congestion out of proportion to the hepatization. The exudation within the air spaces is composed of fibrin, pus and serum. The mucous membrane of the bronchi is congested and swollen.

The catarrhal gastritis is of ordinary type.

The colitis may involve the lower part or the upper part of the colon. There is an increased production of mucus, or an exudation of serum.

The kidneys may be the seat of acute degeneration, or of acute exudative nephritis.

In any one case either one or several of these inflammations may be present.

Symptoms.—In most of the infectious diseases, besides the fever and constitutional symptoms due to the toxin, some one part of the body is regularly inflamed.

In influenza any part of the body, or several parts at once or successively, may be inflamed, or the constitutional symptoms are present without any inflammation at all. Still further than this the most important of all the inflammations—the pneumonia—is not due to the influenza bacillus, but to an additional infection with pneumococci.

The ordinary forms of the disease are as follows:

1. Cases characterized by fever alone. These patients complain little, or not at all, of headache or of pains, nor do they have symptoms referable to any of the viscera. But every day the temperature ranges between 99° and 103° F., lower in the morning, higher in the evening. Some of the patients are confined to bed, some remain in the house, some continue at their work, although they feel miserably. The fever lasts a few days, or even two or three weeks.

In the cases that only last a week the diagnosis is easy. Those that continue for two or three weeks resemble typhoid fever so closely that the diagnosis is difficult. All the cases I have seen were in private practice in consultation so that I have no good histories. It is, however, often to be seen.

2. Cases characterized by pain in addition to the fever. The pain may be a frontal headache, sometimes of great severity; or the pain may be referred to the back and limbs; it may follow the course of any of the nerves. It may be localized in the chest as if due to pleurisy, pneumonia, pericarditis or angina pectoris. It may be referred to some point in the abdomen as if due to a localized peritonitis.

There are cases with severe pains in groups of muscles and complete inability to use these muscles, in which the inflammation may be either in the sheaths of the nerves or in the muscles.

The rise of temperature is rapid, within twelve hours it runs up to from 102° to 106°, but it soon falls and the later temperatures are not much over 101°. The frequency of the pulse and breathing rises and falls with the temperature.

CASE I. *Influenza with fever and headache.* Male, 35 years old, Jan. 19, 1901.

This afternoon chill, temperature 102°, pulse 96, breathing 24, headache, pains in the back and legs, no catarrhal inflammations or other symptoms.

Second day: Temperature 102°—100, headache much better.

Third day: Temperature 100°—99.6°. Urine 1034, no albumen, hyaline casts.

Fifth day: Convalescent.

CASE II. *Influenza with fever and headache.* Male, 23 years old, Dec. 31, 1897.

Four days ago he began to have headache, pain in the back, fever and prostration. There was no coryza, no cough, no abdominal pain.

Fifth day: Temperature 104°, pulse 104, breathing 24, tongue coated and dry, severe headache, urine normal.

After the eighth day the temperature was not above 100°. The headache continued with diminishing severity until the twelfth day. He was not entirely well until the sixteenth day.

CASE III. *Figure* 1. *Influenza with fever, headache and pain in the chest.* Male, 16 years old, Dec. 21, 1896.

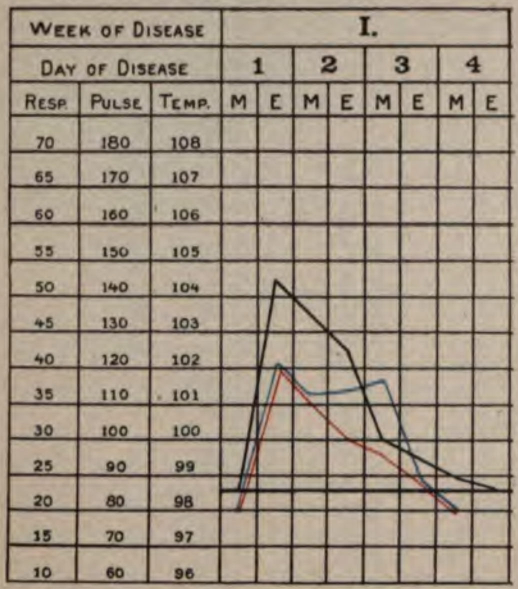
This afternoon began to have chill, fever, headache and pain in the right side of the chest. There were no physical signs over the heart or lungs. Urine 1020, no albumen.

The disease ran its course without any other symptoms and terminated on the seventh day.

CASE IV. *Influenza with mild fever and severe pain in the chest, like angina.* Male, 45 years old, Jan. 27, 1898. Four hours ago he was suddenly attacked with severe pain confined to the præcordial region and moderate prostration, no other symptoms.

Now, temperature 100°, pulse 94, breathing 24. The breathing is rather feeble, without rales. The heart's action is regular, not strong, no murmur.

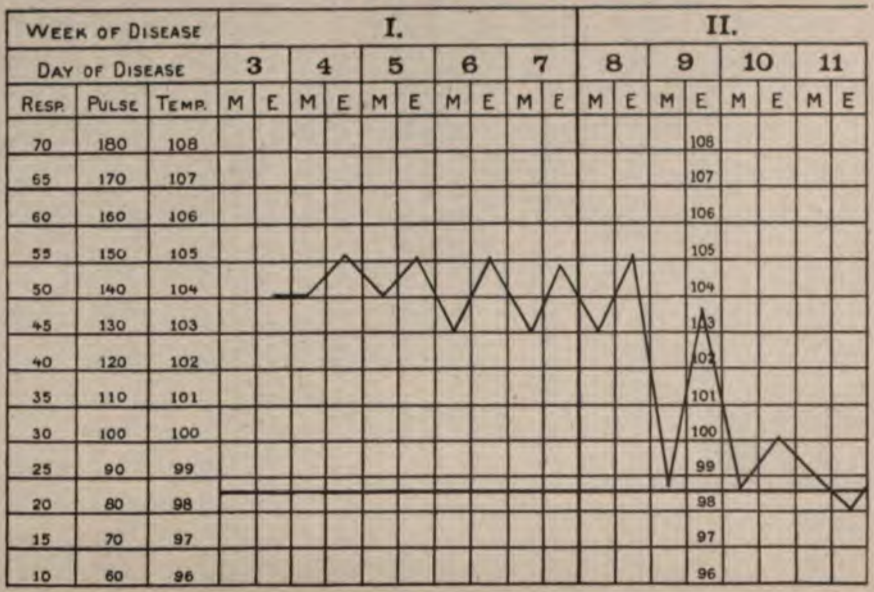
FIG. 1



Temperature black, Pulse red, Respiration blue.

CASE III.—INFLUENZA.

FIG. 2.



CASE VII.—INFLUENZA.

The chest pain continued for three days and then disappeared. The temperature fell to the normal and he was well.

CASE V. *Influenza with severe abdominal pain without inflammation.* Female, 44 years old. Dec. 10, 1897.

She was perfectly well until two weeks ago, then she caught cold and had a dry cough and soreness across the chest. She kept at her work and got nearly well.

Four days ago she suddenly had severe pain in the right iliac fossa. The pain was continuous, worse on deep inspiration; so severe she had to go to bed. Since then she has been feverish. Yesterday she vomited. The bowels were constipated, but have been moved twice by laxatives. She has had a bad headache.

Fifth day: Temperature 103°, pulse 122, breathing 28. Urine 1010, trace of albumen with red and white blood cells. There was marked pain and tenderness with slight muscular rigidity in the right iliac region. She was seen by Dr. McBurney, who made out no appendicitis.

The pain continued until the seventh day. The temperature fell to the normal on the ninth day, and she was entirely well.

3. Cases characterized by great bodily and mental prostration in addition to the fever. In these patients, although the bodily depression is distressing, the mental depression is much worse. It approaches melancholia, and may even pass into it. The fever usually only lasts a few days, but the mental and bodily depression may continue for weeks or months.

Some of these patients become insane, some never get back to their previous condition of good health. Some have fresh attacks of influenza winter after winter so that they do not get the chance to recover.

4. Cases characterized by acute catarrhal inflammations of the conjunctiva, nose, pharynx, tonsils or larynx, in addition to the fever. In these patients there are the ordinary local symptoms which belong to the inflammations of these mucous membranes in addition to the fever and prostration of the influenza. The disease and the inflammations run their course inside of a week.

CASE VI. *Influenza with fever, conjunctivitis, coryza and pharyngitis,* seven days. Male, 34 years old, Jan 8, 1898.

Twenty-four hours ago began to have chills, conjunctivitis, coryza and pharyngitis, with headache, nausea and general pains.

Second day: Temperature 102°, conjunctivitis, coryza pharyngitis, headache, general pains, prostration.

Seventh day: Temperature has fallen to the normal, the catarrhal inflammations are well.

CASE VII. *Figure 2. Influenza with laryngitis.* Female two years old. Dec. 29, 1898.

Two days ago, without evident cause, she became irritable lost her appetite, coughed, had general pains, nausea and vomiting.

Third day: She looked quite sick and had a dry laryngeal cough. There were no physical signs over the heart or lungs. The temperature chart begins on the third day.

She was treated for the laryngitis with hot sponges to the throat and the wine of antimony.

Fifth day: The laryngitis is better, she is less irritable and takes her food although the temperature is still high.

The temperature did not fall until the ninth day, although she continued to improve.

After the eleventh day the temperature was normal.

She was discharged on the sixteenth day apparently well but still very fretful.

CASE VIII. *Influenza with pharyngitis.* Male, 35 years old, March 23, 1898.

Four days ago he began to have chills, body pains, headache, coryza, sore throat, nose bleed, vomiting and diarrhœa.

Fifth day: Temperature 106°, tongue dry, pharyngitis urine 1.018, some albumen with hyaline and granular casts.

Sixth day: Temperature 98° to 103°.

Eleventh day: Temperature fell to the normal, convalescent.

5. Cases characterized by bronchitis, which may be developed at any time in the course of the influenza, or even after it has subsided.

In New York bronchitis is one of the ordinary winter inflammations (bronchitis, page 139). It has been found within a few years that the influenza bacillus is present in the sputum of nearly every case of bronchitis. And yet we see some patients who seem to have a bronchitis and nothing else. We see others who, besides the bronchitis have the fever, headache, general pain and prostration of influenza. It is not easy to say how closely these two sets of cases are related to each other. The problem is farther complicated by the fact that the identification of the influenza bacillus is not as certain as it might be

CASE IX. *Influenza with bronchitis.* Male, 27 years old Dec. 25, 1898.

This afternoon he was suddenly taken with chill, headache, vomiting and severe pain in the left side.

Now, temperature 102°, pulse 116, breathing 28, prostration, headache, cough with muco-purulent, blood-stained sputum. There are fine and coarse rales over the left lung behind. The urine is normal.

Second day: Chill followed by rise of temperature to 106° In the afternoon the temperature fell to 100°, with sweating.

Fourth day: He feels much better, the cough is better with but little sputum; the temperature is normal.

Sixth day: He seems to be well.

6. Cases characterized by pneumonia. In children the type is that of broncho-pneumonia in adults that of lobar pneumonia. The inflammation of the lung may be developed at any time in the course of the influenza, or after it has subsided. In adults the pneumonia involves one or more lobes, with complete or incomplete hepatization, often with congestion out of proportion to the extent of hepatization. In nearly all the cases there is marked catarrhal bronchitis of both lungs. The invasion is either like that of an ordinary lobar pneumonia, or more gradual. The physical signs are those of the bronchitis and of the consolidation. The course of the temperature is not like that of an ordinary pneumonia; there is no sudden defervescence, and the fever sometimes continues after resolution has taken place. The expectoration is usually very profuse, more like that of bronchitis than like that of pneumonia. There is in

many cases a marked disposition to general venous congestion, with failure of the heart's action. The duration of these pneumonias is often longer than that of ordinary lobar pneumonia, and the resolution slower.

My own experience with the influenza pneumonias of New York, both clinical and pathological, has given me the conviction that they are all due to a double infection. In the cases called influenza pneumonia there are four separate conditions either one of which may predominate: The toxin of influenza, the toxin of the pneumococcus, the bronchitis and the inflammation of the lung.

CASE X. *Influenza complicated with pneumonia, the symptoms of the influenza predominating over those of the pneumonia.* Male, 29 years old, Jan. 21, 1898.

One week ago he began to have severe pains all over the body, slight cough with scanty sputum, no chills, no pain in the side, no coryzza, no conjunctivitis, nausea and vomiting, severe headache.

Seventh day, temperature 104°, pulse 100, breathing 28, no dyspnœa, moderate prostration. Urine 1.018, no albumen.

Over the right chest behind, below the angle of the scapula, there is slight dulness on percussion, and diminished breathing. Over the rest of the right lung there is rough breathing and sonorous rales. Severe headache.

Eighth day, temperature 104° to 102°, pulse 100 to 118, actively delirious.

Ninth day, temperature 103° to 101°, actively delirious.

Tenth day, temperature 102° to 98°, delirious.

Eleventh day, temperature 99° to 98°, delirious.

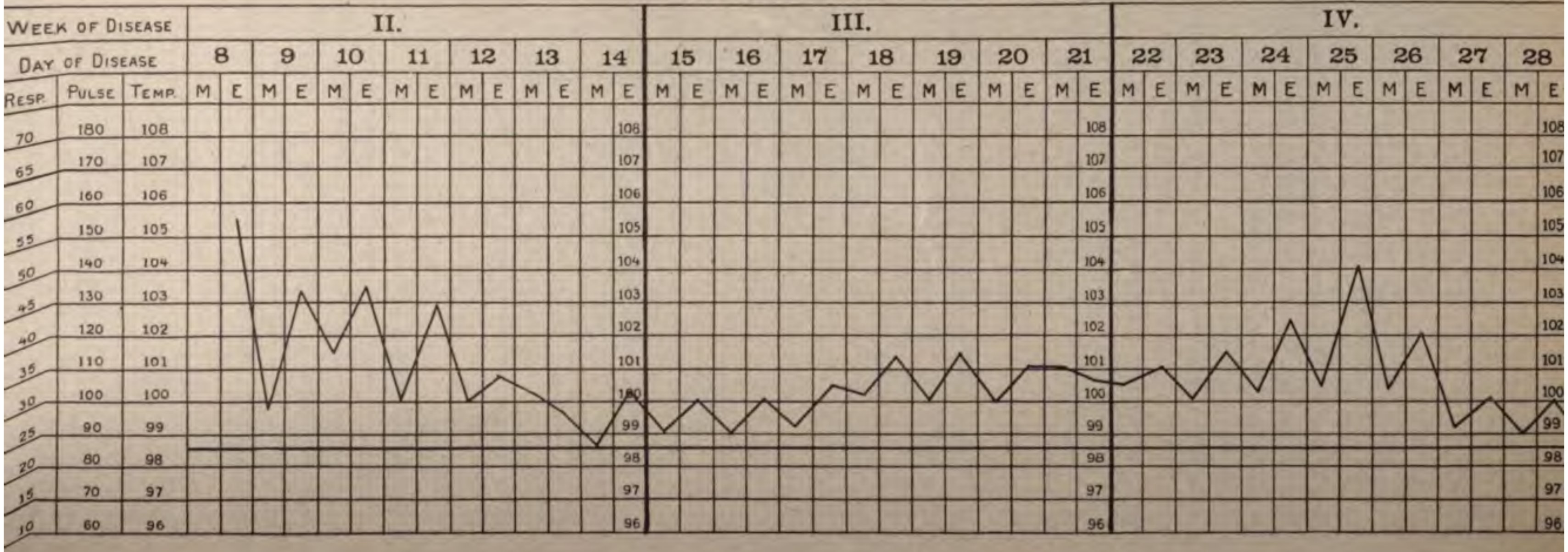
Twelfth day, temperature 100° to 98°, begins to sleep and is becoming rational.

The temperature did not fall to the normal till the eighteenth day.

See pneumonia, page 205.

7. Cases characterized by inflammation of the pleura. Dry pleurisies, involving a part or the whole of the pleura on one side of the chest, are not uncommon. They occur at any time in the course of the influenza. They are accompanied with

FIG 3.



CASE XII.—INFLUENZA.

pleuritic friction sounds, more or less pain, and a moderate rise of temperature. Pleurisies with the production of fibrin and serum, or with fibrin, serum, and pus, are usually not primary, but follow a lobar pneumonia.

8. Cases characterized by acute catarrhal gastritis. The patients suffer from gastric pain, nausea, vomiting and irritability of the stomach, occasionally with vomiting of blood. These symptoms last only for a few days, or are continued for several weeks. In the protracted cases the emaciation and prostration are extreme and death may follow.

9. Cases characterized by acute catarrhal colitis.

Besides the fever and prostation of the influenza, there may be a colitis of the upper or lower part of the colon. If the upper part is involved, there is more or less abdominal pain with large, watery passages. If the lower part is inflamed there are small, frequent, painful passages of mucus and blood. Usually the inflammation runs its course within two weeks and the patients are not at any time very sick.

Case XI. *Influenza with acute catarrhal colitis.* Male, 46 years old, April 10, 1897.

Two days ago he began to have chills, fever, headache, vomiting and general pains.

Third day, temperature 106°, tongue coated and moist, urine 1.022, no albumen. He has frequent, small, painful passages of mucus and blood.

Fourth day, temperature 104° to 98°, after this the temperature was not over 101°.

Thirteenth day, the colitis has run its course, the temperature has fallen to the normal, the patient is convalescent.

Case XII. Figure 3. *Influenza for four weeks with fever, headache, body pains, pharyngitis, bronchitis, colitis.* Female, 20 years old, Feb. 15, 1897.

One week ago she began to have pains in the soles of both feet without redness, or swelling. Then she had headache, pain in the back, thighs and chest and cough with yellow sputum. She has also had several loose movements every day, but has managed to keep at her work.

Eighth day, face flushed, tongue coated and moist, moderate prostration, both thighs tender, urine 1.012, no albumen. There is diminished breathing over the lungs, and a systolic heart murmur in the second left space.

Twelfth day, she vomits from time to time, the sputum is bloody, the bowels continue to be loose with serum and mucus.

Seventeenth day, the cough, bloody sputum and diarrhœa have stopped; she still vomits, but is evidently better.

Thirtieth day, she is convalescent.

10. Cases characterized by a variety of rashes in the skin.

11. Cases which behave like acute cerebro-spinal meningitis. The patients have a febrile movement which runs an irregular course. They are much prostrated and look very ill. They have the ordinary cerebral symptoms: headache, photophobia, pain, tenderness, and contraction of the muscles of the back of the neck, vomiting, delirium, stupor, general convulsions.

12. The cases with inflammation of the sheaths of the nerves suffer from a good deal of pain along the course of the nerves, followed by a loss of power and atrophy of the muscles.

There is a good deal of variety in these cases as to the duration of the pain and the difficulty in distinguishing between neuritis and myositis.

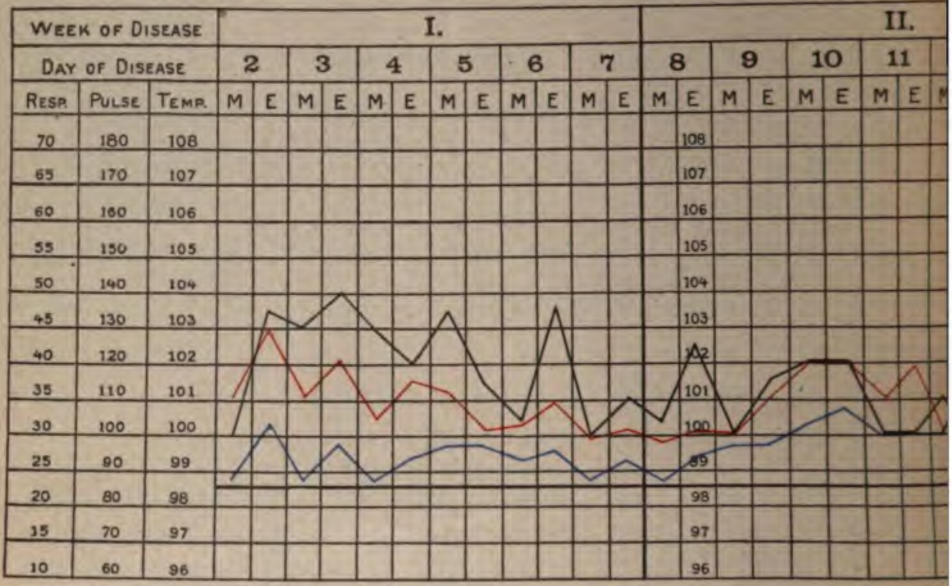
Case XIII. *Influenza with pain in the muscles.* Male, 23 years old, Jan. 12, 1897.

Five days ago he began to have pain in both thighs and the back so severe that he could not walk. The pain was so severe that he could hardly move, but otherwise he did not feel very sick.

Sixth day, temperature 103°, pulse 118, urine 1.030, no albumen. The tongue is coated, there is moderate prostration, he cannot move his legs on account of the pain.

Ninth day, there is no more pain in the legs, the temperature has fallen to the normal.

13. In a fair number of cases of influenza there is a temporary albuminuria with acute degeneration of the kidneys. In



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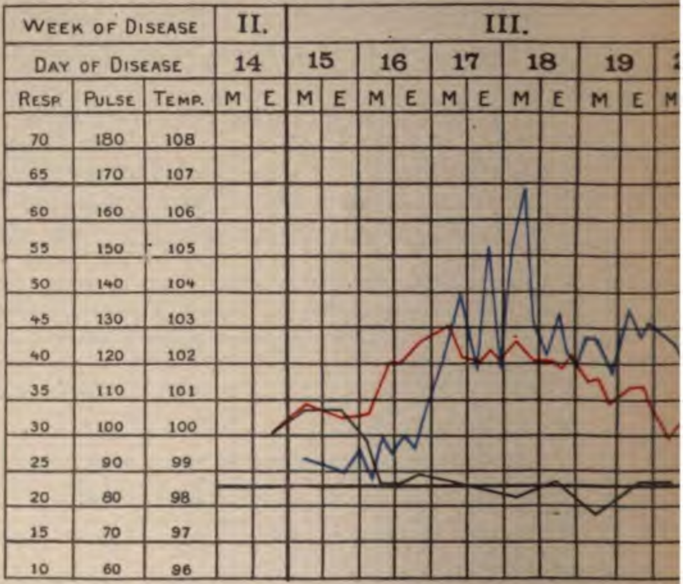

Temperature black, Pulse red, Respiration blue.

Case XV.—Influenza.

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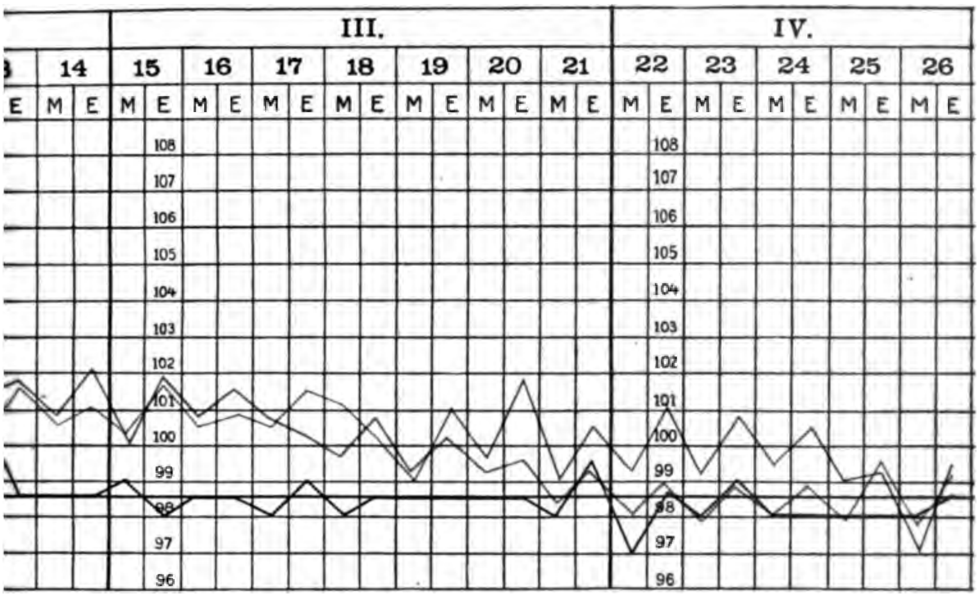


ed. Respiration blue.

FLUENZA.

a moderate number of cases there is an acute exudative nephritis. (Kidney, page 445.)

14. Tachycardia is a prominent feature in a few cases of influenza. It begins during the influenza and may be continued for weeks or months afterward. It is accompanied with abnormal cardiac sensations and mental depression. The worst cases I have seen were in men already poisoned with tobacco.

15. During the winters of 1903-1904 I saw several cases characterized by rapid pulse, rapid breathing and great prostration, one of them died. The rapid pulse and breathing came on later in the influenza, sometimes with œdema of the lungs.

CASE XIV. Figure 4. *Influenza with disturbed heart action, rapid breathing and oedema of the lungs.* Female, 40 years old, Nov. 18, 1903.

She began then with fever, headache, prostration and a dry cough.

Nov. 22. I saw her for the first time. The noticeable things were the rapid and labored breathing, the rapid and exaggerated heart action, the tympanitic distension of the abdomen and the extreme prostration. The physical signs were those of œdema of the lungs.

After Nov. 30, she had no fever, but the bad breathing and prostration continued.

Dec. 4. The bad breathing and bad heart action with the distension of the abdomen continue; she looks very ill.

Dec. 8. There is a moderate improvement and she can eat a little solid food.

Dec. 20. She has continued to improve and to-day is moved on a couch.

Jan. 11. She is convalescent.

CASE XV. Figure 5. *Influenza with rapid breathing and heart action; sudden death.* Male, 58 years old, Nov. 10, 1903.

Two days ago he began to have pain in the left side, which continued and increased.

Nov. 10. No cough, no dyspnœa, no fever, no headache. Does not feel sick, but has the pain in the left side. There is a friction sound over the lower lobe of the left lung.

Nov. 11. Eyes suffused, temperature 100°, moderate prostration, friction sound continues.

Nov. 12. Temperature 102.6°.

Nov. 13. Feels much better, is out of bed and eating.

Nov. 18. Temperature 100°, not as well, friction sound continues.

Nov. 21. The chart begins. There is a friction sound all over both lungs.

Nov. 22. He feels very miserable, with dyspnœa, palpitation, friction sound over both lungs.

Nov. 23. During the previous night the dyspnœa became worse and the palpitation more annoying. There was profuse perspiration.

During the day he vomited several times, was unable to lie down on account of the dyspnœa, face pale and dusky. He became very much depressed and expected to die. In the evening he complained of great agony, asked to be killed, and tried to get out of the room. There was some œdema of the lungs.

Nov. 24. He complains bitterly of agony and pain, but does not wish for death. The dyspnœa and palpitation continue.

Nov. 25. He is decidedly better, although the breathing continues to be very rapid. In the middle of the day he sat up and ate some lunch. But the breathing is so bad he cannot lie down or sleep. Urine 1.030, a trace of albumen.

Nov. 26. He seems better, except for the breathing and pulse. At 11 P. M. he went to bed and slept all night.

Nov. 27. He was very sleepy all day and really looks as if he were getting better. He slept most of the time until 9 P. M.

At 10 P. M., he suddenly looked worse and in ten minutes was dead.

CASE XVI. *Influenza fatal without lesions.* Male, 34 years old, Jan. 28, 1890.

On Jan. 24 and 25, he felt unwell, but continued to work.

Jan. 26, he had headache, vertigo, pains in the chest, stomach and back, vomiting, coryza, prostration.

Jan. 28, temperature 104°, pulse 88, breathing 18, urine 1.010, no albumen, vomited twice with a little blood, some cough with mucous sputum.

Jan. 29, temperature 103° to 104°, pulse 88 to 90, breathing 20-24.

Jan. 30, temperature 103° to 104°, pulse 88-86, breathing 24.

Jan. 31, temperature 103° to 102°, pulse 90 to 92, breathing 28 to 26, vomiting.

Feb. 1, temperature 101° to 102°, pulse 88 to 96, breathing 26, vomiting.

Feb. 2, temperature 101° to 102°, pulse 88 to 96, breathing 26.

Feb. 3, temperature 102° to 103°, pulse 92 to 96, breathing 28.

Feb. 4, temperature 100° to 102°, pulse 82 to 90, breathing 26, seems to be better.

Feb. 5, temperature 101°, pulse 84, breathing 28. He looked better and was given a chop for dinner, but vomited in the afternoon.

Feb. 6, in the morning he felt better, and was placed in a chair while his bed was made. It was soon noticed that he felt badly, became pale, the radial pulse disappeared, he vomited, passed his feces and died.

Feb. 7. Autopsy.

The brain and its membranes were normal. The heart was rather small, there was old thickening of the aortic and mitral valves.

The lungs were only partly inflated; all the capillaries contained red blood cells, but were not distended; the cells which line the air spaces were much swollen but not detached; there was no exudate.

The liver was congested.

The spleen was large.

The stomach and intestines were normal.

The kidneys were congested.

In all these different forms of influenza, after the disease has run its course, the patient may return to his natural health within a short time. But one of the distressing features of the disease in many cases has been the mental and bodily depression, which continues for weeks and months after the acute symptoms have subsided.

Dr. Hutchings, of the St. Lawrence State Hospital, gives an analysis of forty cases of post influenzal insanity. There were nineteen men and twenty-one women. The percentage of hereditary cases was 26.8 for men and 47.6 for women. Melancholia was the form of the disease in 18, dementia in 6, general paralysis in 5, and mania in 9. Of the men 6 recovered, 4 were improved, 3 died, 2 were discharged unimproved, and 3 became chronic. Of the women 8 recovered, 3 were improved, 2 were discharged unimproved, 3 died, and 5 remained under treatment.

Treatment.—For the influenza as such there is no treatment.

The headache, if dependent on inflammation of the frontal sinuses, can be relieved in some cases by local applications to the nose. For the general headache and for the pains in other parts of the body, the different coal tar preparations are of service.

The various accessory inflammations are to be treated as is usually done.

Case I.—Influenza with fever and headache.

Case II.—Influenza with fever and headache.

Case III.—Figure 1. Influenza with fever, headache and pain in the chest.

Case IV.—Influenza with fever and pain in the chest like angina.

Case V.—Influenza with severe abdominal pain.

Case VI.—Influenza with fever and catarrhal inflammation.

Case VII.—Figure 2. Influenza with laryngitis.

Case VIII.—Influenza with pharyngitis.

Case IX.—Influenza with bronchitis.

Case X.—Influenza with pneumonia.

Case XI.—Influenza with acute catarrhal colitis.

Case XII.—Figure 3. Influenza for four weeks with fever, headache, pharyngitis, bronchitis and colitis.

Case XIII.—Influenza with pain in the muscles.

Case XIV.—Figure 4. Influenza with rapid breathing and heart action.

Case XV.—Figure 5. Influenza with rapid breathing and heart action, sudden death.

Case XVI.—Influenza, sudden death without lesions.

RHEUMATISM.

Any persons who have pain in the joints or muscles are popularly said to have rheumatism.

From this whole group we can separate the cases of gout, of gonorrhœa, of scarletina, of rheumatoid arthritis, of puerperal rheumatism and some of the cases of chronic rheumatism.

Inflammation of the muscles, muscular rheumatism, seems to be due to a variety of causes.

There remains a group of cases that may be mild or severe, run a subacute, acute or chronic course and yet behave as if they were all caused by the same poison, a poison that is mild or virulent, of short duration or continued for long periods.

Concerning the nature of the poison we are ignorant, but its effects are, within certain limits, regular. It causes: fever, evidences of poisoning, inflammations, loss of nutrition, secondary anæmia, rarely death. The poison in the majority of cases ceases to cause symptoms after three weeks, but its duration may be much longer, and the same person may suffer from repeated attacks at short or long intervals.

The disease is most common in the temperate zones, but is also observed in cold and warm climates.

The time of greatest liability to the disease is between the ages of ten and thirty years. There seems to be both an hereditary and a personal predisposition to the disease. The same individual often suffers from several attacks. The strong and vigorous and the feeble and debilitated seem to be equally liable to the disease. Exposure to cold and wet often act as exciting causes.

Symptoms.

1. The ordinary cases of acute articular rheumatism with toxæmia, fever and inflammation of the joints.

The invasion of the disease is usually in one of three ways:

(a) For a few hours, or for several days the patients complain of general malaise, headache, irregular chills, a little fever, irritability, sleeplessness, a coated tongue, no appetite, urine loaded with urates and irregular pain about the joints.

(b) There are pains and stiffness of one or more joints, gradually increasing and followed after some hours, or days, by fever.

(c) The invasion is sudden with chills, a rapid rise of temperature, and inflammation of one or more joints.

When the disease is established there is a rise of temperature usually between 101° and 104°. This fever may not last for more than two or three days, more frequently it continues for a week, sometimes longer. Thus in 100 ordinary hospital cases I found the duration of the fever to be one week or less in 37 cases, and inside of two weeks in 68 cases. But there are cases in which the fever lasts for 5 or 6 weeks. When the fever lasts longer than this it is due to repeated attacks of the disease.

With the fever the pulse is full and rapid, the skin is hot and dry or bathed in perspiration, the tongue is coated, nausea and vomiting are often present, the bowels are constipated, the urine is diminished in quantity and loaded with urates, the intelligence is clear, but restlessness and sleeplessness are apt to be marked features.

One or more joints are inflamed, swollen, painful and tender. It is an acute synovitis with congestion and an exudation of serum. In protracted cases the fluid becomes purulent, or the capsule of the joint thickened. There are cases, however, in which pain is the only evidence of inflammation of the joint. The cases vary as to the intensity of the synovitis, the severity of the pain, the number of joints inflamed and the way in which one joint after another is involved.

The inflammation of the joints regularly lasts longer than the fever and may be followed by a chronic synovitis. The following table gives an idea of the ordinary relative duration of the fever and the synovitis:

TABLE OF THE COMPARATIVE DURATION OF THE INFLAMMATION OF THE JOINTS AND THE FEVER, BASED ON 200 CASES FOR THE JOINTS, 100 CASES FOR THE FEVER.

	Joints.		*Fever.*	
Duration.	No. Cases.	Percentage.	No. Cases.	Percentage.
1 week	5	2½	37	37
2 weeks	22	11	31	31
3 weeks	28	14	14	14
4 weeks	16	8	4	4
5 weeks	28	14	7	7
6 weeks	21	10½	4	4
7 weeks	11	5½	1	1
8 weeks	11	5½	1	1
9 weeks	8	4	0	0
10 weeks	6	3	1	1
11 weeks	6	3		
12 weeks	6	3		
13 weeks	6	3		
14 weeks	3	1½		
15 weeks	3	1½		
16 weeks	2	1		
17 weeks	2	1		
18 weeks	1	½		
19 weeks	2	1		
20 weeks	2	1		
21 weeks	3	1½		
22 weeks	1	½		
23 weeks to year	7	3½		

CASE I. *Acute articular rheumatism, ordinary mild case, seven days duration.* Male, 21 years old, Dec. 30, 1898.

He had an attack of rheumatism four years ago.

Two days ago he began to have coryza and a cough with muco-purulent sputum. That evening the right knee became swollen, tender and painful. The next morning the left knee was inflamed.

Third day, temperature 102°, pulse 104. Urine 1.008, no albumen, no physical signs over the heart or lungs. He was put on alkaline treatment.

Seventh day, the temperature has fallen to the normal and none of the joints are inflamed.

CASE II. *Articular rheumatism protracted, fever for 36 days, joints still inflamed at the end of 62 days, treatment without effect on the disease.* Female, 29 years old, May 18, 1899.

On May 14, she began to have pain, tenderness, redness and swelling in the right knee, then in the left knee, the ankles and the right hand. At the same time she had chills, fever, sweating, vomiting and sore throat.

There were no physical signs over the heart and lungs.

The temperature ran between 100° and 103° up to the 23d day, between 99° and 101° to the 32d day, but did not get down to normal till the 36th day.

The inflammation of the joints continued, sometimes better, sometimes worse. On the 62d day there was still some pain and tenderness.

She took at different times sodium salicylate, alkalies and antipyrine. The joints were treated with ice, oil of wintergreen, blisters and iodine. It was evident that the treatment had no effect either on the constitutional symptoms or the inflammation of the joints.

CASE III. *Acute articular rheumatism, protracted, aspiration of knee joint, incision of sterno-clavicular joint.* Male, 46 years old, Jan. 9, 1899.

No previous attacks of rheumatism. For several weeks he has had some pain and stiffness in the joints. One week ago he began to have headache, fever, profuse perspiration. The next day the left knee became swollen, red and painful.

Now, temperature 101°, pulse 100, left knee swollen, red and painful, urine 1.032, no albumen, no physical signs of the heart, or lungs.

He was put on alkaline treatment with the inflamed knee wrapped in oil of wintergreen.

Jan. 14, the temperature has been running between 100° and 102°, he is restless and delirious, the left knee continues to be a good deal swollen.

Jan. 15, the knee was aspirated and two ounces of turbid serum withdrawn. The left sterno-clavicular articulation has become red, swollen and tender.

Jan. 18, the temperature runs between 99° and 100°, the knee is almost well.

Feb. 2, the man is nearly well, except for the inflammation of the sterno-clavicular articulation. This was on Feb. 2d incised and packed. There was no pus in the joint, but the capsule was infiltrated and thickened. After this he improved steadily; by Feb. 9th there was no more fever; Feb. 23d he was well.

Treatment.—The very mild cases of articular rheumatism unquestionably get well with simple rest in bed without any treatment. On the other hand, there are some cases that are not benefited by any treatment. The majority of cases are helped by treatment.

The routine treatment in New York is with salicylate of soda in doses of 10 to 20 grains every two or three hours. The objection to this is the number of patients who suffer from salicylic poisoning. For this reason aspirin, salicin, or the oil of wintergreen, are often used with equally good results.

There are a moderate number of cases that seem to do better with large doses of alkalies.

There is a group of cases characterized by excessive pain in the joints without much evidence of inflammation. These do better with phenacetine, acetanilid, antipyrine, or salol.

If there is much distension of the knee joints with fluid it is wise to aspirate them.

2. Ordinary cases of rheumatism complicated with iritis, tonsilitis, pericarditis, endocarditis, pleurisy or pneumonia.

Iritis is fortunately rare. It is apt to be painful and troublesome.

Tonsilitis adds nothing to the disease except the pain and swelling of the throat.

The combinations of pericarditis and endocarditis with rheumatism are described on pages 325 and 333.

The pleurisy is either with fibrin alone, or with fibrin and serum.

The pneumonia is a lobar pneumonia of the pneumococcus type. It is not easy to be sure of the exact relationship of the rheumatism and the pneumonia.

CASE IV. *Acute articular rheumatism, pericarditis, endocarditis, pleurisy with effusion.* Male, 15 years old, Jan. 19, 1899.

No previous attacks of rheumatism. Five days ago he began to have pain in breathing. Soon there was pain and swelling of the left ankle, and fever.

Sixth day, temperature 101°, pulse 118, breathing, 28. He complains of a good deal of pain over the heart, the left ankle is swollen and tender, both knees are painful. Urine 1.022, no albumen. There is a double friction sound all over the front of the heart and a loud systolic murmur at the apex; the action is regular and rapid.

He was put on the salicylate of soda.

Eleventh day, the temperature has been running between 101° and 103°, the pulse between 104 and 128. To-day the temperature has fallen to 99° and the pulse to 80, the precordial pain and dyspnœa have gone.

He continued to do well until the twenty-second day, then the temperature ran up to 102°, and the next day there was fluid in the left chest.

Twenty-fourth day, 150 c.c. of clear serum was aspirated from the left chest.

Twenty-seventh day, 250 c.c. of clear serum was aspirated from the left chest.

Thirty-fourth day, the temperature has fallen to the normal he is well. He was not strong enough to leave the hospital until the forty-third day.

3. Cases of acute articular rheumatism with repeated attacks at short or long intervals.

When the attacks are repeated at intervals of one or more years, each attack behaves like an ordinary attack and is to be treated in the same way. The principal danger is that of fresh endocarditis with progressive damage of the endocardium.

When the attacks are repeated at intervals of days or weeks the matter is much more serious. The disease is practically continuous, the nutrition suffers, a marked secondary anæmia is established. The ordinary drugs for rheumatism cease to be of any value, and our only resource is the use of the natural hot and sulphur baths. 635

4. Cases of acute articular rheumatism with endocarditis, without inflammation of the joints.

There are a certain number of persons in whom we find an endocardial murmur, disturbed heart action, a secondary anæmia, increased leucocytosis, loss of nutrition and perhaps a little rise of temperature. It is evident that they all have endocarditis. In how many of them the poison of rheumatism is the cause of the endocarditis it is not so easy to say.

A considerable number of them do very well indeed on combinations of iodide of potash and iodide of iron.

5. Cases of acute articular rheumatism with well marked inflammation of the joints, but very little fever.

These patients are not very sick and do well on the ordinary drugs for rheumatism.

CASE V. *Articular rheumatism, well marked inflammation of the joints, hardly any fever.* Female, 39 years old, May 2, 1896.

She has already had several attacks of rheumatism. Four days ago the knees and shoulders became swollen and painful, later the finger joints were inflamed. There was headache and loss of appetite, but no fever, or very little. Now, temperature 99°, pulse 100, urine 1.010, albumen and granular casts. No physical signs over the heart or lungs. The shoulders, knees and fingers were inflamed, swollen and painful. She was given salicylate of soda and iodide of potash.

After this the temperature was not over 99°, the inflammation of the joints subsided and by May 8 she was well.

6. Cases with secondary anæmia, loss of nutrition, little or no fever and very little inflammation of the joints.

These are very bad cases. They continue for months and years. Treatment is very unsatisfactory. The ordinary drugs for rheumatism are of little service. The natural mineral baths, massage, an out-of-door life in a suitable climate, the use of iron and of fats seem to be the best we can do.

7. Arthritis after rheumatism. In a certain number of cases the poison of rheumatism runs its course and disappears, but one or more joints—the hands, knees, or ankles—remain inflamed. Either the cavity of the joint is distended with seropurulent fluid, or the capsules and surrounding tissues are thickened. Besides the impairment of movement there is often very severe pain.

Treatment.—In managing these cases it is better to forget all about the rheumatism and only treat the arthritis. If there is fluid in the joint it must be aspirated. For the pain and thickening pressure, rest, extension, passive motion are to be employed as necessary. I think surgeons manage these cases better than physicians do.

CASE VI. *Arthritis after rheumatism.* Female, 20 years old, March 13, 1900.

She had one attack of rheumatism twelve years ago.

Two days ago she began to have pain and stiffness in the feet, shoulders and elbows, with headache, chills and fever.

March 13, temperature 102°, pulse 118, both knee joints much swollen, ankles and wrists swollen and tender, no physical signs over the heart and lungs. She is put on the salicylate of soda.

March 16. There is no improvement in the joints.

March 19. The other joints are better, but the right knee is tense and painful. An ice cap is applied to it.

March 30. The right knee continues to be swollen and painful, otherwise she is much better. The salicylate of soda is stopped.

April 12. Hæmoglobin 50 per cent., red cells 2,688,000, white cells 8,200. The swollen right knee was aspirated, but no fluid obtained. After this the knee was blistered and baked. It continued to be swollen and painful until July 4, then it was put up in Buck's extension. It eventually got entirely well.

CASE VII. *Acute articular rheumatism, one knee, ineffectual treatment; the joint not well after two months.* Female, 30 years old, Sept. 22, 1896.

Several years ago she had rheumatism in both knees.

Twenty-four hours ago the left knee became swollen and painful, she had headache and feeling of heat and cold. Now, temperature 101°, pulse 92, the left knee is swollen, hot and very tender. There are no physical signs over the heart or lungs. She was given salicylate of soda, the knee was put on a posterior splint, poulticed and bandaged.

Sept. 24, an ice cap was put on the knee.

Oct. 3, the knee was blistered.

Oct. 18, the knee was poulticed.

Nov. 7, posterior splint and bandage.

Nov. 27, walked with crutches.

Dec. 6, she was discharged from the hospital with the knee still painful and tender.

CASE VIII. *Arthritis after rheumatism.* Male, 56 years old, Jan. 30, 1900.

He has had two attacks of rheumatism a number of years ago.

Five days ago he began with chills, fever, headache and prostration.

The next day the right knee became painful and also the right shoulder. Now, temperature 100°, pulse 88, urine 1.027, albumen and granular casts. The right knee is swollen and painful. He was put on the salicylate of soda and the knee wrapped in oil of wintergreen and cotton batting.

Feb. 14. I saw him for the first time. The temperature has been running between 99° and 101°, except on Feb. 11, when for a few hours it went up to 103°. He continued to take salicylate of soda, the knee continued to be very painful but was left undisturbed in the cotton batting.

I found the knee very much swollen and distended with fluid. On Feb. 14, one ounce of turbid fluid was aspirated; on the 15th two ounces more were aspirated. Feb. 17 he was transferred to the surgeons. They made a thorough aspiration of the knee and washed it out with boric acid.

After this he recovered with good use of the knee.

9. The virulent cases.

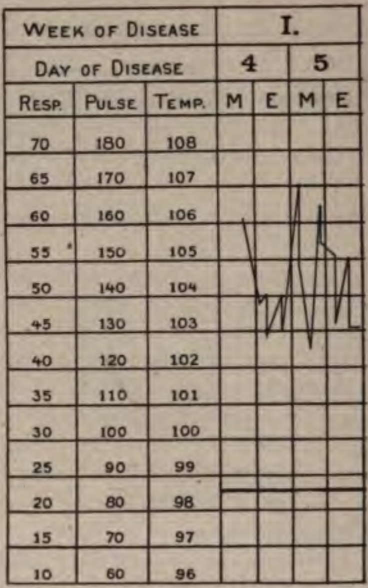
In rheumatism, as in the other infectious diseases, the poison in a moderate number of cases is virulent and capable of causing death without complications.

The marked features of these cases are the unusually high temperature and the evidences of a virulent poison. The tongue becomes brown and dry; the pulse is rapid and feeble, the breathing is rapid, the patients suffer from restlessness, sleeplessness, hyperæsthesia of the skin, muscular twitchings, general convulsions, delirium and coma. Some of them die in a few days, a few live for several weeks, recovery is possible.

Treatment.—The ordinary treatment for rheumatism in these cases does nothing. The best results obtained so far have been with tub bathing.

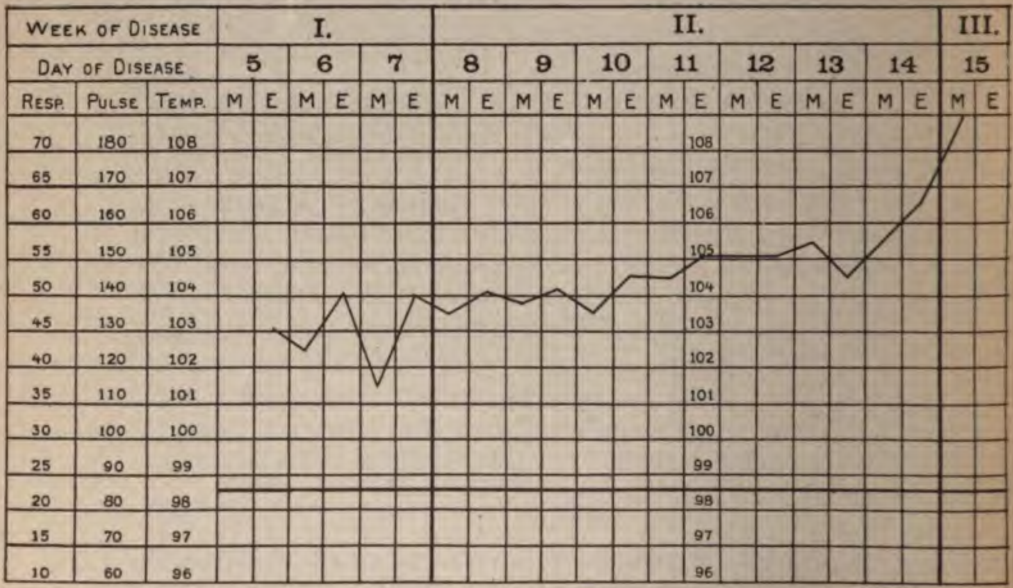
CASE IX. Figure 1. *Virulent rheumatism, death on the fifth day.* Male, 40 years old, Jan. 22, 1899.

FIG. 1



CASE IX.—RHEUMATISM.

FIG. 2.



CASE X.—RHEUMATISM.

Jan. 19. He began to have pain in the abdomen and back, headache, loss of appetite, high fever, profuse perspiration, a feeling of stiffness in the joints, which developed into severe pain by the next morning in the right hand and shoulder and the left knee.

Fourth day, temperature 106°, pulse 108, tongue moist and coated, right hand and left knee tender, but not swollen. Urine 1.024, considerable albumen. No physical signs over the heart or lungs. He was given three tub baths at 65°, which reduced the temperature to 104°.

Fifth day, temperature 107°, pulse rapid, weak and irregular, no delirium, very little pain in the joints. He had three more tub baths, which reduced the temperature, but did no good. He became first delirious, then stupid, the pulse more rapid and feeble, and at 9 P. M. he died.

The autopsy showed no lesions.

Case X. Figure 2. *Acute articular rheumatism, fatal. Invasion with endocarditis.* Female, 25 years old, May 12, 1898.

One month ago she began to have headache, dyspnœa on exertion, palpitation, loss of appetite and a pallor that gradually increased. These symptoms seem to mark the beginning of the endocarditis.

Two weeks ago her throat became sore and the headaches more severe. Four days ago both knees, the ankles and shoulders became swollen, tender and painful.

May 12, the temperature chart begins. Both knees are inflamed. The heart's action is rapid and forcible, there is a mitral systolic murmur. She was given salicin.

May 17, the joints are not much inflamed, but the patient is doing badly; there is a double friction sound over the front of the heart. She was given alkalies in large doses.

She continued to have high temperature, to lose flesh and strength, became delirious, and finally died on May 22, apparently from the rheumatism alone.

Case XI. Figure 3. *Virulent form of acute articular rheumatism, death on twenty-fifth day.* Male, 50 years old.

April 12, he began to have a severe attack of articular rheumatism with marked inflammation of the large joints. The temperature chart begins on that day. He was treated with large doses of salicylate of soda.

April 24. Hæmoglobin, 64 per cent., red cells, 3,000,000, white cells 18,920.

April 26. I first saw him. He was then very ill and badly poisoned, dry tongue, delirious, the joints still inflamed but not much swollen. There were no physical signs over the heart or lungs. The liver was large and cirrhotic. I thought he would probably die.

May 1. Hæmoglobin 58 per cent., red cells 3,180,000, white cells, 17,215.

May 2, I saw him the second time. The inflammation of the joints had subsided, but he was doing badly in every other way.

May 7 he died.

10. Chronic rheumatism.

Lesions.—A chronic inflammation beginning in the synovial membrane of one or more joints. The inflamed membrane becomes dense and thick, the fluid within the synovial sac is usually scanty and grumous, sometimes abundant and serous. The inflammation may extend to the capsules and ligaments of the joints, the articular cartilages, and the bones.

As complicating lesions we find chronic endocarditis, chronic endarteritis, pulmonary emphysema and bronchitis, and chronic nephritis.

Causes.—The disease belongs to adult life and to old age. It is especially common among the poor and among persons who are constantly exposed to all sorts of weather. In some persons the course of the chronic rheumatism is varied by attacks of acute rheumatism.

Symptoms.—The symptoms are principally the local symptoms of the inflamed joints. But from time to time there may be a little rise of temperature, and, after a while, digestive disturbances, loss of nutrition, and the symptoms belonging to the complicating lesions are developed.

The inflammation begins in one or more joints, often those of the hands and feet, continuing in these joints and then gradually extends to others, until in the worst cases nearly all the joints in the body are involved. The affected joints are

tender and painful, swollen, sometimes fluctuating, sometimes stiff, sometimes creaking. The course of the inflammation is slow, with remissions and exacerbations.

The patients vary as to the position and number of the joints inflamed, and as to the extent of the changes in the joints.

The prognosis is always bad, but yet in some patients the disease never cripples them completely.

The only efficient treatment consists in the use of the natural mineral waters taken internally and as baths. The best waters seem to be those of Sharon, Richfield, Virginia, North Carolina, Arkansas, Wiesbaden and Aix-les-Bains.

Case I.—Acute articular rheumatism, ordinary mild case.

Case II.—Acute articular rheumatism, protracted.

Case III.—Acute articular rheumatism, aspiration of knee joints.

Case IV.—Acute articular rheumatism with pericarditis, endocarditis and pleurisy with effusion.

Case V.—Acute articular rheumatism with well marked inflammation of the joints, but very little fever.

Case VI.—Arthritis after rheumatism.

Case VII.—Arthritis after rheumatism.

Case VIII.—Arthritis after rheumatism.

Case IX.—Figure 1. Acute articular rheumatism, virulent form, death on the fifth day.

Case X.—Figure 2. Acute articular rheumatism, virulent form.

Case XI.—Figure 3. Acute articular rheumatism, virulent form.

THE IRREGULAR FEVERS OF NEW YORK.

We see in New York a large number of sick persons in whom a rise of temperature is the principal symptom.

In a considerable number of these cases there is a recognizable definite cause for the fever so we may call such cases regular fevers.

Under this head come:

1. Typhoid fever. This is always present in New York. If the infection is severe the course of the disease is fairly typical. If the infection is mild the disease runs a more or less irregular course. But yet the diagnosis can usually be made.

2. Malarial fever of the tertian form with regular paroxysms of chills, fever and sweating is not uncommon, especially in the northern portions of Manhattan Island. It is to be noted, however, that blood taken from the skin often fails to show the presence of malarial organisms.

3. Influenza. Some rise of temperature belongs to all the cases of influenza but is regularly associated with headache or one of the accompanying inflammations. There are cases, however, in which a fever lasting for one, two or three weeks is the only symptom.

4. In the simple anæmia of young women and in pernicious anæmia a rise of temperature in the afternoon is regularly present. As a rule the more profound the anæmia the higher is the temperature.

5. Sepsis. When we say a person is septic we mean that he is poisoned with the toxin of one of the pathogenic bacteria. The ordinary bacteria are: streptococci, pneumococci, staphylo-

cocci, gonococci, and the bacillus coli communis. With such poisoning there can be any variety of fever, or even no fever at all. And yet when we see a temperature curve running sharply up and down from 97° to 105°, or more, with chills before the rise of temperature it regularly means sepsis. The number of leucocytes is sometimes increased, sometimes not.

6. Tuberculosis. Nearly all tubercular inflammations are attended with a rise of temperature. The height of the temperature is in proportion to the virulence of the infection rather than to the extent of the inflammation. The cases with a small tubercular focus and fever as the principal evidence of the infection are sometimes puzzling.

7. Syphilis. Attention has recently been called to continued fever as a late symptom of syphilis. Some of the cases reported are straightforward, others will hardly bear criticism.

8. Endocarditis. It is now well known that there are cases of endocarditis without disturbances of circulation, but with fever as the prominent symptom. If the fever continues for months with very few heart symptoms the cases may be very obscure.

9. Ptomaine poisoning with its attendent fever is of short duration and easily made out.

On the other hand we also see a number of sick persons in whom a rise of temperature is the principal symptom, but we are unable to determine the cause.

These we call cases of irregular fever. Under this head come:

1. Irregular fevers which suggest the idea of malarial poisoning:

(a) Fevers which run a short course of from one to four weeks.

The fever is an afternoon fever of from 99° to 102°, exceptionally 105° to 107°. The morning temperature approaches the normal.

The patients have more or less headache and feeling of illness so that they are more comfortable in bed than out of it, but yet they do not look very sick. There is loss of appetite and there may be disturbances of the stomach and bowels. The spleen is not enlarged, nor are there any physical signs. The blood shows no changes except those of a moderate secondary anæmia.

Quinine by itself has no effect on the fever.

All of the patients recover. In some the fever simply runs its course, in some arsenic and quinine together seem to control it, some do not get well until they are taken out of New York.

Case I. Figure 1. *Irregular fever of nine days duration resembling malaria.* Male, 28 years old, on the evening of October 27th had a chill, followed by fever.

October 28, the temperature chart begins. He has no symptoms except the fever, loss of appetite and general malaise.

Oct. 29. His condition is the same.

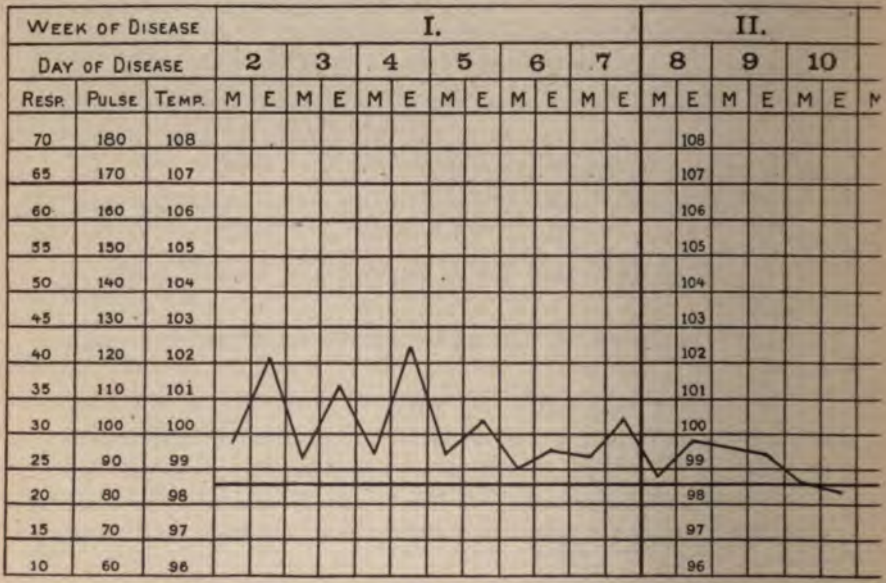
Oct. 30. He feels better, can eat and goes out in an automobile.

Oct. 31. Urine 1.005, no albumen or casts. He is given arsenic, quinine and acetanilid together.

Nov. 1. He is distinctly better.

Nov. 4. He is well and remained so.

FIG. 1.



Case I.—Irregular Fever.

CASE II. *Irregular fever of malarial type for twenty-eight days.* Male, 45 years old, Nov. 21, 1902.

For 10 days he has been feeling miserable with an afternoon fever and headache. He has taken large doses of quinine without any benefit.

He now looks sick, but not much prostrated; temperature at 12 M., 101°.

Nov. 25. He came back to the office, not improved; afternoon temperatures, 100° to 102°. He was given arsenic, quinine and acetanilid together.

Nov. 29. The fever continues, he is no better.

Dec. 1. The lungs, heart, liver and spleen are normal. Urine, a trace of albumen and a few granular casts. Widal reaction negative, no malarial organisms in the blood.

Dec. 3. The afternoon temperature is still 102°.

Dec. 9. He is well and remained so.

CASE III. Figure 2. *Irregular fever resembling malaria.* Female, 35 years old, Nov. 12, 1902.

She has lived for some time at Yonkers, and has just moved to New York. For the past three months she has had irregular chills and fever. The attacks have varied in severity, duration and frequency, but have never lasted more than two days. She has taken quinine.

One week ago she had a chill in the night followed by fever. She went to bed and was better after two days.

Nov. 11, she had a chill followed by fever, she feels miserable most of the time.

Nov. 12. She is well nourished, hæmoglobin 60 per cent., no malarial organisms. She is given arsenic, quinine and iron in combination.

Nov. 14. The temperature chart begins. The lungs and heart are normal, the liver and spleen are not enlarged. Urine 1.018, no albumen or casts.

Nov. 16. She begins to feel better.

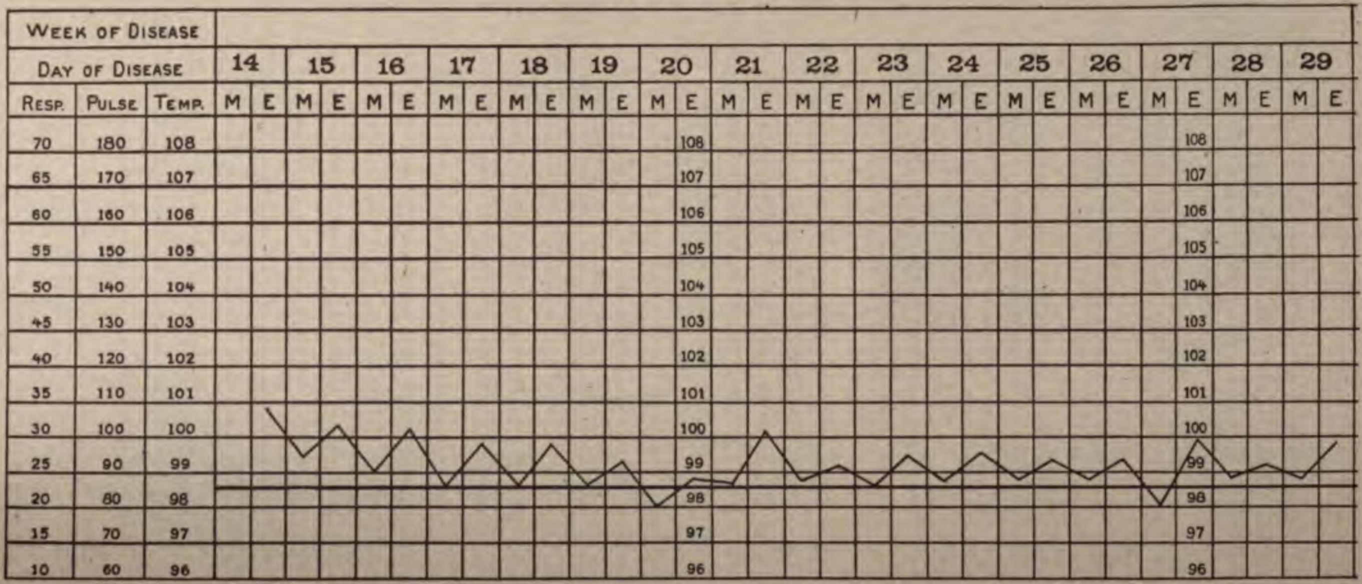
Nov. 21. She feels well, hæmoglobin 70 per cent.

After December 7th there was no more fever. It will be seen that before that time the afternoon temperatures hardly reached 100° and yet during all that time every afternoon she was more or less miserable.

Dec. 1. Hæmoglobin 66 per cent., she is given iron alone.

Dec. 9. She continues to grow stronger, so that she can do shopping without fatigue.

FIG. 2.



CASE III.—IRREGULAR FEVER.

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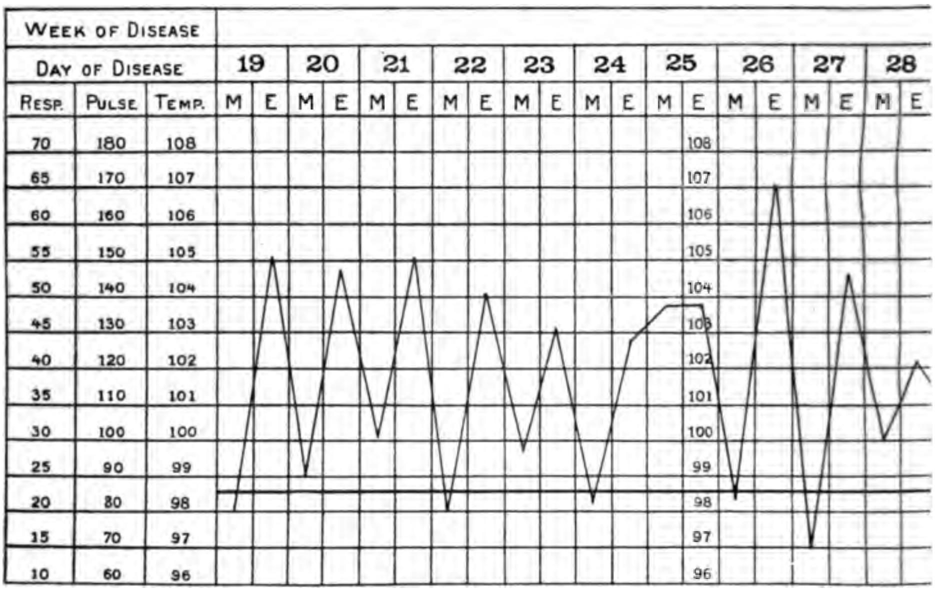
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CASE IV.—

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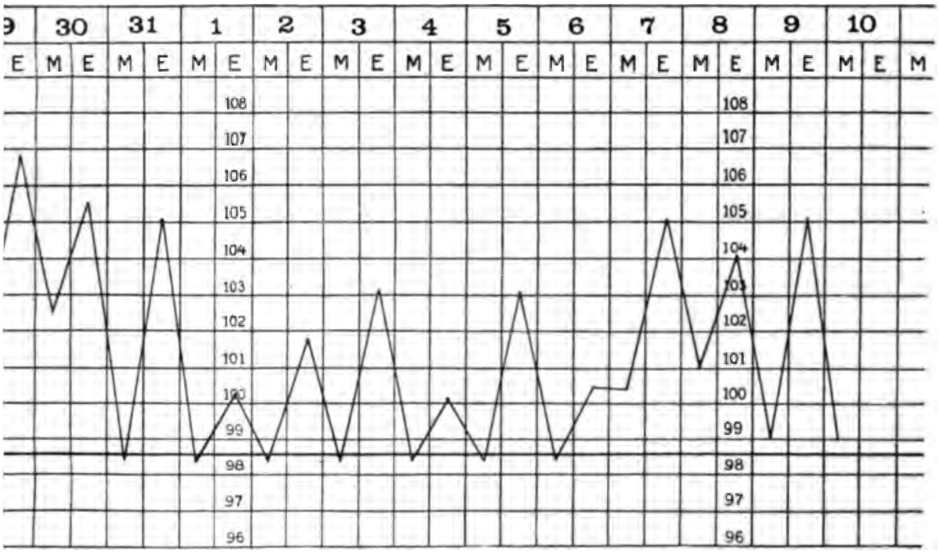

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ULAR FEVER.

Dec. 22. She now feels perfectly well and strong, hæmoglobin 70 per cent., all medicines stopped.

This is a good example of the patients who first have distinct malarial paroxysms followed by a continuous afternoon fever, which may last for several weeks. Although the morning temperatures are normal and the afternoon temperatures not over 100°, the patients continue to feel miserable until the fever has entirely gone.

CASE IV. Figure 3. *Irregular fever of unusual type without discoverable cause, recovery.* Female, 12 years old, about May 15th began to have irregular fever with pains in the head.

May 17. The drum of one ear was incised and nothing found.

May 19. The temperature chart begins.

May 24. The mastoid of the same ear was opened and nothing found.

She took large doses of quinine most of the time without result.

She did not at any time even with the highest temperatures look septic or seriously ill.

On June 10th she was put in the cars and taken to Canada. After four days in camp she had no more fever, was well and remained so.

(b) Irregular fevers of long duration. I have seen this in children and adults, in persons living in many different parts of the city. The morning temperatures are normal. The afternoon and evening temperatures are from 99° to 100°, and the pulse is then fuller than at other times.

In the morning the patients feel well, adults attend to their business, children go to school or play.

In the afternoon they feel depressed and miserable, they have no appetite for their evening meal, they do not sleep well during the first part of the night. The symptoms are so indefinite as to puzzle both the patients and the physicians. For one or two weeks the patients often think they are merely tired, or run down. But when for two or three months they find that every afternoon brings its misery they become discouraged. There is no limit to the duration of this afternoon fever. It may continue steadily for months, it may stop for months and then begin again; it may come back winter after winter. Many persons only have it while they are in New York, a few are worse when they go to the country. It is very capricious. It is possible to have it one winter and never again, or for two or more winters and then it will stop and not return.

Drugs have no effect at all on this fever. Either it stops of itself, or because the patient changes his residence to another part of New York, or leaves the city altogether.

2. Irregular fevers with a distinct septic curve of temperature, but no discoverable focus during life. In some of these cases a purulent focus is discovered after death. Others recover and no focus is ever made out.

I put these cases together because clinically they run the same course. Whether there is really a form of sepsis without a purulent focus I do not know.

These cases last for a long while—many weeks. They are usually seen by a number of physicians and various diagnoses are made. It has seemed to me that it is not generally understood that this curve of temperature regularly means sepsis whether we can find the focus or not.

CASE V. Figure 4. *Septic fever for 234 days with a small sacculated empyema, which was not discovered until the autopsy.* Female, 47 years old, June 29, 1898.

She then had the ordinary symptoms of rheumatoid arthritis.

July 8. The temperature began to rise in the afternoon.

July 13. There was some pain in the left chest, but no physical signs.

November. She began to have a cough, with occasional bloody sputum, no physical signs.

Jan. 11, 1899. She had an abscess in the left heel. The cough was worse and the sputum more profuse, no tubercle bacilli.

During all the long time she was in the hospital no one was able to get any physical signs or to make a diagnosis. She was worse and better with the rises and falls of temperature. She lost flesh and strength and died Feb. 26, 1899.

Autopsy. The body was emaciated.

Near the root of the left lung is a small sacculated empyema, around this a thin layer of lung is necrotic. Both lungs are emphysematous and the bronchi contain muco-pus.

The heart is normal, except for an adherent pericardium.

The stomach and upper part of the duodenum are dilated.

The liver, spleen and kidneys are normal.

CASE VI. Figure 5. *Death from sepsis with purulent inflammation of the middle ear and right shoulder.* Male, 40 years old. March 19, 1900.

Seven days ago he began with chill, headache, prostration, no vomiting.

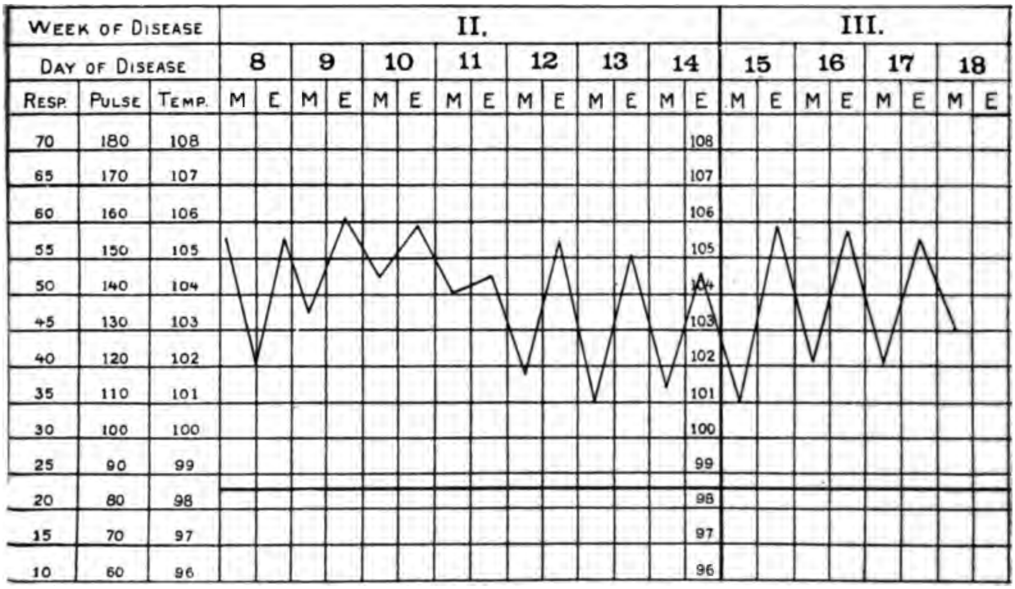
Five days ago he became delirious.

Two days ago another chill and pain in the right leg.

Now, the tongue is brown and dry, the abdomen is tympanitic, the man is delirious and septic.

March 20. A chill for ten minutes in the afternoon, delirium continues.

FIG. 5.



CASE VI.—IRREGULAR FEVER. FATAL SEPTIC FEVER.

March 22. The evidences of general sepsis continue, the right leg is swollen.

March 23. The right shoulder is swollen and tender.

March 30. He died, thoroughly septic.

Autopsy. The brain and its membranes were normal.

The left middle ear contained pus.

The right shoulder joint contained pus.

The lungs were emphysematous.

The heart, kidneys, liver and stomach were normal.

In this case the sepsis was entirely out of proportion to the ear lesion.

CASE VII. Figure 6. *Irregular fever of septic type lasting from May 5 to July 15, possibly due to an abscess near the rectum.* Male, 45 years old.

About May 1 began to have the fever, which is seen in the chart. There were no other symptoms except a gradual loss of flesh and strength. On many days the rises of temperature were preceded by chills.

He was seen by a number of physicians. No one could find evidence of any local disease. There was no increased leucocytosis, no malarial organisms. A variety of diagnoses were made. I saw him on May 24. I could not make out any evidence of a local lesion in any part of the body. Although he was feeble and emaciated he did not look so very badly. I thought he was suffering from septic poisoning without a discoverable focus and advised to give no medicine, but to begin to feed him and get him out of bed.

This was done and in about a week he was strong enough to be moved to a hotel in the suburbs. There he continued to gain flesh and strength in spite of the continuance of the fever.

Finally from July 15th to 18th he passed every day from the rectum 8 to 12 stools composed principally of pus. After that he had no more fever and gained rapidly.

I heard a year later that he was quite well and attending to his business.

CASE VIII. Figure 7. *Irregular fever of eighty days duration, septic type, no focus, recovery.* Female, 32 years old, Dec. 5, 1897.

She was perfectly well until 16 days ago. Then she began to have abdominal pain low down on the right side. The next day she had a severe chill, followed by fever. The pain has

continued, the bowels have been loose, there has been a cough with muco-purulent sputum. No headache, epistaxis, hæmoptyses, night sweats or vaginal discharge.

Now she complains of pains all over the body and weakness. The tongue is heavily coated, moderate dyspnœa, some abdominal tenderness in the right iliac region, moderate tympanites, no eruption, urine 1.010, a little albumen, many pus cells. There are no physical signs over the chest or abdomen. She looks seriously ill.

Dec. 7. There are no tubercle bacilli in the sputum.

Dec. 23. A distinct chill, followed by sweating. No malarial organisms are found in the blood.

Jan. 13. Thrombosis of left femoral vein.

About the 65th day she began to improve and got entirely well.

CASE IX. Figure 8. *Irregular fever of septic type for eighty-five days, recovery.* Male, 23 years old, Jan. 28, 1901.

Two weeks ago he caught cold.

Eleven days ago he woke up in the early morning with a severe chill and sharp pain in the upper part of the left chest. Since that time he has been in bed, growing weaker with persistent cough and pain in the left chest.

Now, the tongue is brown, dry and coated. The respiration is rapid, shallow and labored. The abdomen is normal. Both knees and the left shoulder are stiff and painful. The cough is infrequent with scanty rusty sputum. He is very weak, pulse 88, breathing 44. Urine 1.026, trace of albumen, no casts. Dulness over the base of the right lung without change in the voice or breathing, no physical signs over the heart, liver, or spleen.

Jan. 29. Tongue dry and brown, no cough or sputum, no pain in the chest, hæmoglobin 80 per cent., red cells 4,704,000, white cells 10,800.

Jan. 30. Tongue moist and coated, feels better and stronger, abdomen a little distended.

Feb. 3. Tongue brown and dry, a good deal of pain in the shoulders and right arm.

Feb. 7. The tongue continues dry, he is somewhat apathetic, he has a good deal of pain in the shoulders and chest. Hæmoglobin 90 per cent., red cells 4,784,000, white cells 16,800. He does not improve.

Feb. 11. The tongue is clean and moist, he has no more pain, but looks sick.

Feb. 20. He is beginning to improve and is a little hungry.

Feb. 27. He sits up in bed.

Feb. 28. He sits up out of bed.

March 12. He is steadily improving, hæmoglobin 75 per cent, red cells 3,861,000, white cells 6,000.

March 14. Urine 1.016, a good deal of albumen, many pus cells and granular casts.

March 19. Urine 1.016, trace of albumen, pus cells, no tubercle bacilli. He continues to gain flesh and strength.

March 23. Hæmoglobin 75 per cent. red cells 4,400,000, white cells 14,200.

April 1. He is out of bed, the urine contains very few pus cells.

April 24. He is well and in very good condition.

3. There are long irregular fevers with marked cerebral symptoms that may terminate in death or recovery and yet there is no meningitis. Some of the best examples of this form I have seen in consultation and have no histories.

Case X. *Irregular fever with cerebral symptoms for sixteen days, death, no lesions.* Female, 20 years old, March 29, 1882.

Ten days ago she began to have chills, loss of appetite, nausea and vomiting.

The next day the face, arms and legs were covered with a rash, severe headache and photophobia, the bowels have not moved for three days.

Now, the tongue is dry, the left pupil larger than the right; headache; red, tender, elevated patches on the arms, legs and face. Urine 1.025, a trace of albumen. Temperature 103°, pulse 100.

March 30. Temperature 103° to 103°, pulse 96 to 116.

March 31. Temperature 101° to 102°, pulse 100. She cries out, makes faces and is unable to swallow.

June 1. Temperature 101° to 99°, pulse 96 to 88.

June 2. Temperature 102° to 100°, pulse 96.

June 3. Temperature 95°, skin cold, pupils dilated; she died.

Autopsy.—The pia mater looks a little dry, but shows no inflammatory lesions, the brain is normal.

The spinal cord and its membranes are normal.

The lungs are congested.

The heart is normal.

The liver is large and congested.

The spleen is of medium size and soft.

The pyloric end of the stomach is thickly coated with mucus.

The solitary glands of the ileum are a little swollen.

The mesenteric glands are not swollen.

The kidneys are normal.

The uterus and its appendages are normal.

CASE XI. Figure 9. *Irregular fever with marked cerebral symptoms, recovery.* Female, 25 years old, Oct. 25, 1900.

Four days ago she began to feel chilly and feverish, vomited, felt sick and went to bed.

Now, pale, tongue coated and moist, restless and nervous, no physical signs, urine 1.030, trace of albumen, pulse 148, breathing 40.

Oct. 23. Active noisy delirium with delusions and sleeplessness.

Oct. 26. The sleeplessness and active delirium have continued in spite of bromide, morphine and hyoscine; the tongue is dry; she looks as if she had some infectious disease, pulse 120 to 130, breathing 24 to 32.

Oct. 29. For the first time begins to sleep and is no longer delirious.

Oct. 31. Hæmoglobin 100 per cent, red cells 5,288,000, white cells 9,200; no widal reaction. She still has hallucinations, the tongue has become moist.

Nov. 3. She is still irrational, but is quiet.

Nov. 11. She is now quiet and rational, pulse 80 to 108, hæmoglobin 96 per cent., red cells 5,040,000, white cells 9,800. After this she steadily improved and by Nov. 23d was entirely well. I was unable to make a diagnosis.

4. From time to time we see young women, only moderately anæmic, who for months run an afternoon temperature of about 100°. They are anxious cases, for a miliary tuberculosis without physical signs is always possible. But it is possible to follow these patients for so long a time as to render it probable that no

tuberculosis was ever present. So we have to put these cases under the head of irregular fever without discoverable cause.

5. There are short fevers that resemble influenza and yet seem to be different from it.

CASE XII. Figure 10. *Irregular fever resembling influenza.* Female, 5 years old.

March 13. She came home from dancing school and felt sick and feverish.

March 17. She had some pharyngitis and looked as if she had typhoid fever. She was given three tubs a day at 99°.

After this the temperature fell and the child improved until March 20, when she was well. It was a month before she was all right again.

CASE XIII. Figure 11. *Irregular fever resembling influenza.* Female, 30 years old, Oct. 10, 1900.

Six days ago she began to feel chilly, feverish, with headache, body pains and enough prostration to go to bed. She also had nausea and repeated vomiting.

Oct. 10. She had a severe chill, followed by fever and profuse sweating. She has had a little cough with muco-purulent sputum.

Now, her face is flushed, tongue coated and moist, urine 1.022, a little albumen and a few granular casts.

Oct. 11. She slept very little, vomited occasionally, no malarial organisms were found in the blood.

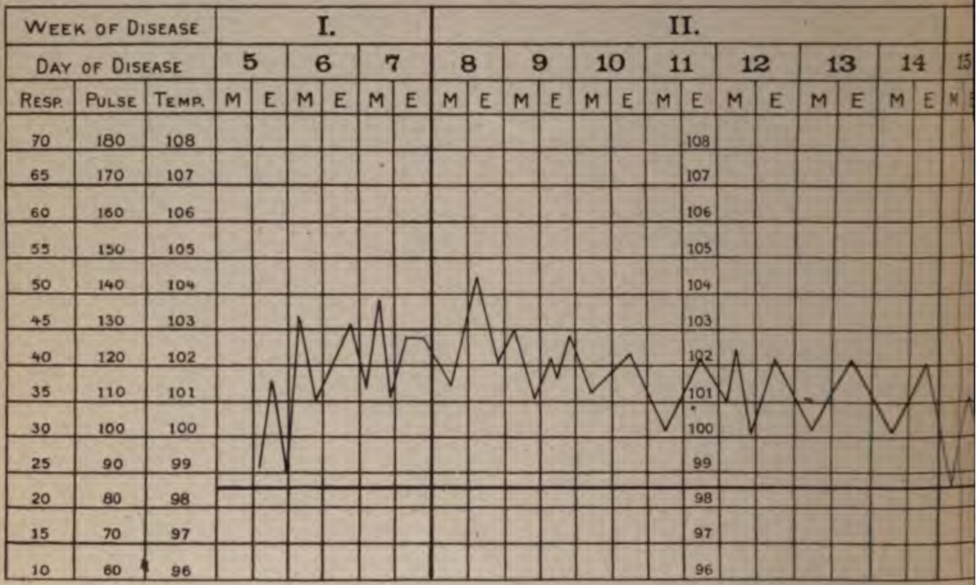
After this she gradually improved and by October 19th was quite well.

6. Irregular fevers of uncertain character. Under this head I can simply describe a few of the cases of severe continued fever for which I was unable to account.

CASE XIV. Figure 12. *Severe continued fever of uncertain origin.* Male, 27 years old, hospital interne.

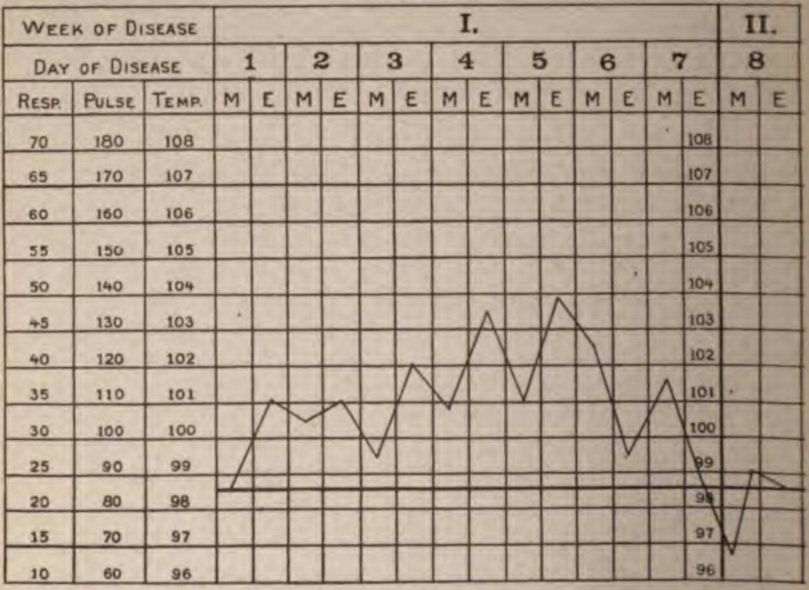
Ten years ago he acquired a gonorrhœa which was not entirely well until the end of ten months. After this there was no stricture and no return of the discharge, but if he drank to excess there was irritability of the neck of the bladder.

At the end of June for a week he drank a good deal.

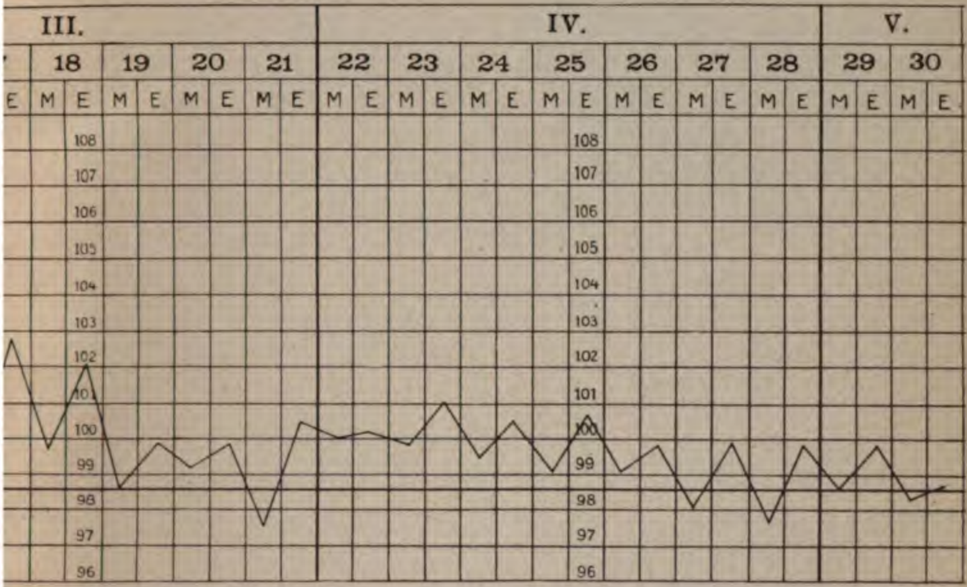


Case XI.—Irregula

FIG.10.

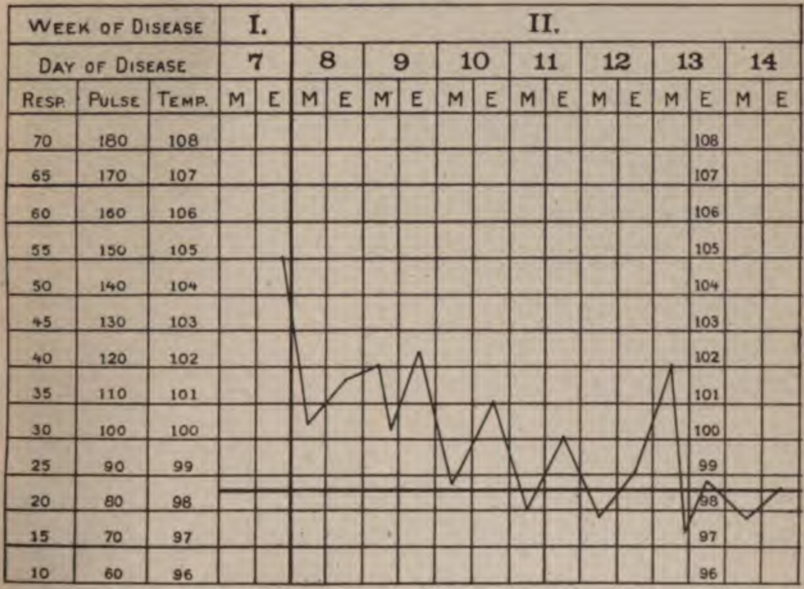


Case XII.—Irregular Fever Resembling Influenza.



EBRAL SYMPTOMS.

FIG.11.



CASE XIII.—IRREGULAR FEVER RESEMBLING INFLUENZA.

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July 1 he went back to work with irritability of the neck of the bladder. For this he was given a deep injection of nitrate of silver. This caused much pain and he had to go to bed, but there was no fever. By the end of July there was no more irritability of the bladder.

August 1 he again went back to work, but soon felt sick—with a temperature of 100° in the morning and 102° in the afternoon, until August 24, when the temperature chart begins. The urine contained a few pus cells, but no gonococci were found at any time.

Aug. 31. An inflamed inguinal gland was cut out. It showed no changes but those of simple inflammation. Leucocytes 8,400, widal reaction negative.

Sept. 3. Night sweats, leucocytes 14,400.

Sept. 5. Three grains of calomel caused salivation.

Sept. 7. He began to have a very severe headache with photophobia, which continued day and night and could not be relieved.

Sept. 15. I saw him for the first time. His condition was that of a man who was very badly poisoned and who was more likely to die than to recover. I thought his only chance was to feed him and take him away from the hospital.

Sept. 18. He was given scraped beef and sat up in a chair for a short time.

Sept. 21. He was carried out into the garden. Although he still has headache and fever he is distinctly better.

Sept. 30. He was taken to his home in the country. After that he had no more headache and continued to improve. The temperature continued at 99° to 100° for three weeks.

Nov. 1. He seemed to be entirely well.

Dec. 1. He went back to the hospital and soon began to feel sick with an afternoon fever.

Jan. 1. He had to give up and went away on a yacht for three months. He did not go back to the hospital.

For two years since he has been perfectly well.

CASE XV. Figure 13. *Irregular fever for fifty days with and after a streptococcus sore throat.* Male, 27 years old, Jan. 18, 1898.

Eight days ago he began to have a dry cough with pain in the throat, fever, headache, abdominal pain and diarrhœa. The throat has continued to be very painful.

Now, there are patches of false membrane over the pharynx and tonsils. There are sibilant and sonorous breathing over the lower lobes of both lungs. Urine 1.013, no albumen or casts.

Jan. 25. No diphtheria bacilli were found in cultures from the throat. The throat is better, but the man continues to be sick.

Jan. 29. Cultures from the throat give only streptococci.

Jan. 31. The throat is clean.

Feb. 3. The man continues to be ill with dry tongue and delirium.

Feb. 9. He is better and hungry.

March 3. He is well.

The point of interest in this case was the long continuance of the fever after the throat was well.

CASE XVI. Figure 14. *Irregular fever for which there was no satisfactory explanation, recovery.* Male, 28 years old, Feb. 15, 1900.

About 17 days ago, after exposure, he began to have prostration, general pains, headache, vomiting, chilliness, fever and had to stop work.

For the first week he was better in the morning and worse in the evening. He felt dizzy, nauseated with occasional vomiting, two or three chills in each twenty-four hours.

During the second week he managed to work in the morning, but was weak and miserable in the afternoons.

Now, his face is flushed, tongue coated. There are no physical signs over the chest and abdomen, hæmoglobin 90 per cent., red cells 5,520,000, white cells 12,000, no malarial organisms were found on repeated examinations. He continued with no symptoms but the irregular fever and prostration until Feb. 22, when he was evidently getting better.

March 1. He seemed to be entirely well.

CASE XVII. Figure 15. *Irregular fever, death, autopsy.* Female, 18 years old, Nov. 28, 1882.

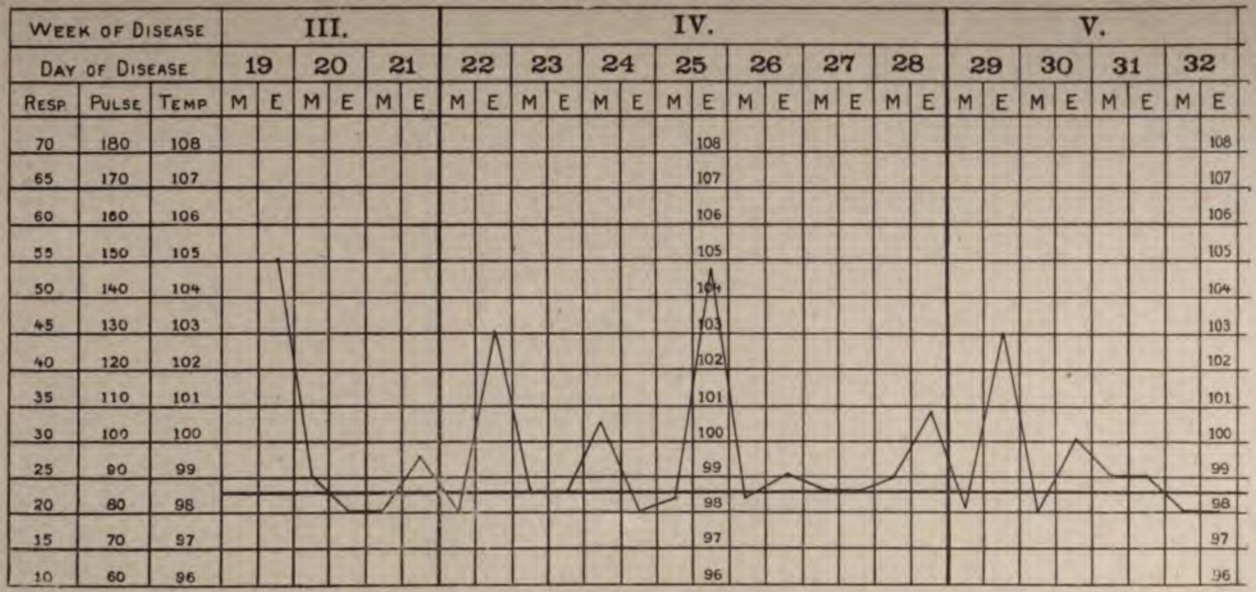
Several years ago she had an attack of chorea which lasted for two months, since that time her health has not been good.

Two years ago she had several attacks of rheumatism.

Now, she feels weak and miserable, pulse 128, breathing 28, a loud systolic murmur at the apex of the heart, urine 1.020, no albumen. She looked feeble, but not very ill.

Dec. 11. She began to menstruate with marked hysterical symptoms. The next day she lost control of the bladder and rectum.

FIG. 14.



CASE XVI.—IRREGULAR FEVER.

She continued to look like an hysterical girl, but gradually grew weaker and more stupid, with a dry tongue.

On January 6th, without other symptoms, the temperature suddenly began to rise and in a few hours she was dead.

Autopsy.—The brain and its membranes showed no changes except a moderate congestion of the pia mater. Minute examination of the pia mater showed no lesions.

In the lungs the large and medium sized bronchi contained muco-pus.

The mitral valve was thickened, moderately stenosed and insufficient.

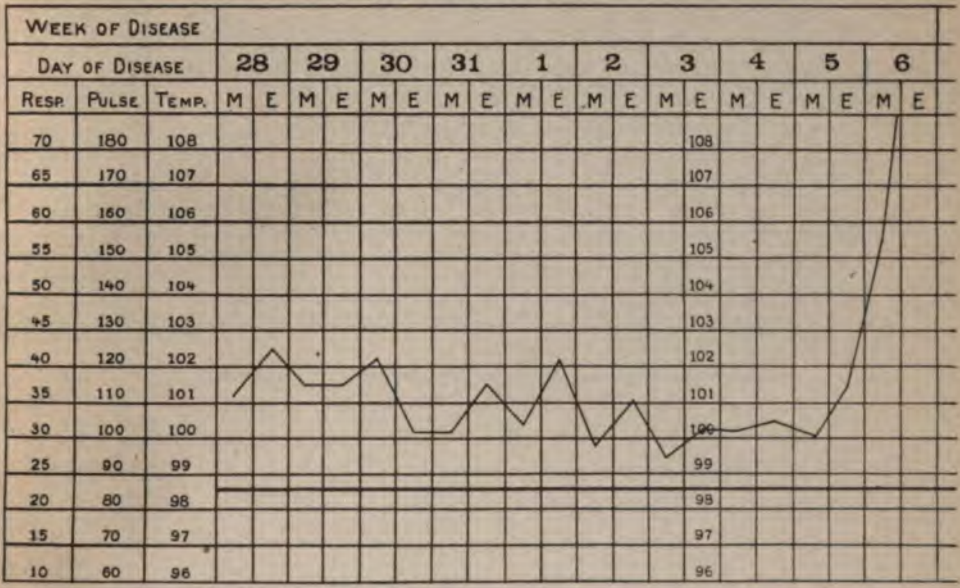
There were no vegetations or roughnesses.

The liver was congested.

The spleen and kidneys were normal.

The stomach, small intestines, uterus and ovaries showed no lesions.

FIG. 15.



CASE XVII.—IRREGULAR FEVER. DEATH.

CASE XVIII. Figure 16. *Irregular fever of obscure character, death, autopsy.* Male, 68 years old, Jan. 17, 1899.

For years he has had attacks of bronchitis and asthma. He has also had several attacks of acute gastritis.

In May, 1898, he began to have dyspnœa on exertion and a feeling of general lassitude.

In October he began to have a cough with muco-purulent sputum and daily attacks of asthma.

Since November 5th the asthma has not been troublesome.

Now, he complains chiefly of cough and weakness. The arteries are not contracted, physical signs of emphysema with subcrepitant rales, good and regular heart action, urine 1.030, no albumen, casts or sugar.

Jan. 18 to 20. There was nausea and vomiting.

Jan. 24. Vomiting and diarrhœa, he is getting more feeble.

Feb. 2. The diarrhœa continues, he is somewhat tympanitic.

Feb. 11. He is delirious.

He looked to me throughout as if he had chronic sepsis.

The House Staff thought he had typhoid fever.

Feb. 16. He died.

Autopsy.—There were patches of fresh fibrin on the pleura. The bronchi contained muco-pus. The lungs were emphysematous, but not congested or œdematous.

The left ventricle of the heart is a little dilated, the muscular fibres are degenerated. The wall of the anterior coronary artery is thickened.

The aorta through its entire length shows patches of old thickening, some of which are covered by thin, fresh thrombi.

The liver is small, in the condition of chronic congestion.

The spleen is normal.

The mucous membrane of the stomach is intensely congested.

The small intestine is normal.

The kidneys, bladder and prostate show no lesions.

Case I.—Figure 1. Irregular fever resembling malaria.

Case II.—Irregular fever resembling malaria.

Case III.—Figure 2. Irregular fever resembling malaria.

Case IV.—Figure 3. Irregular fever.

Case V.—Figure 4. Septic fever for 234 days with a small sacculated empyema which was not discovered until the autopsy.

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Case VI.—Figure 5. Septic fever with purulent inflammation of the middle ear and right shoulder.

Case VII.—Figure 6. Irregular fever of septic type lasting from May 5th to July 15th, possibly due to an abscess near the rectum.

Case VIII.—Figure 7. Irregular fever of septic type for 80 days, recovery.

Case IX.—Figure 8. Irregular fever of septic type for 85 days, recovery.

Case X.—Irregular fever with cerebral symptoms, death.

Case XI.—Figure 9. Irregular fever with cerebral symptoms, recovery.

Case XII.—Figure 10. Irregular fever resembling influenza.

Case XIII.—Figure 11. Irregular fever resembling influenza.

Case XIV.—Figure 12. Severe continued fever of uncertain origin.

Case XV.—Figure 13. Irregular fever for 50 days with and after a streptococcus sore throat.

Case XVI.—Figure 14. Irregular fever.

Case XVII.—Figure 15. Irregular fever.

Case XVIII.—Figure 16. Irregular fever.

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THE LARGE INTESTINE.

The large intestine extends from the termination of the ileum to the anus. It is divided into the cæcum, the ascending colon, the transverse colon, the descending colon, and the rectum.

Its wall is composed of a peritoneal coat, a connective-tissue coat, and a glandular coat. The glandular coat is composed of tubules lined with cylindrical epithelium, which are separated from each other by connective tissue abundantly supplied with blood-vessels. In the wall of the intestine are imbedded numerous small lymphatic glands. It is important to remember the relations of the colon to the vermiform appendix, the liver, and the stomach.

The portions of the food which have not been absorbed in the small intestine pass through the ileo-cæcal valve into the cæcum and then downward through the colon to the rectum. We have very little exact knowledge as to the changes which take place in the food after it passes into the colon. As it comes from the small intestine it is in a semi-fluid condition. As it passes through the colon, water is absorbed from it, so that by the time it reaches the rectum the fæces are comparatively dry and solid. The normal condition of the fæces, therefore, in the ascending colon is semi-fluid, in the rectum nearly solid.

It seems to be evident that the mucous membrane of the colon also excretes solid matters and gases and that in adults a variety of changes take place in the fecal matter.

Our actual knowledge at the present time seems to amount to this:

1. Whatever of food is left over from the small intestine passes into the colon in a semi-fluid state.

2. In a perfectly normal adult this semi-fluid matter loses a certain amount of fluid, has added to it some excrementitious substances and is then passed as solid fæces.

3. In ordinary civilized life the contents of the large intestine often behaves differently.

(a) They may become too hard and dry.

(b) They may undergo a variety of putrefactive changes.

(c) They may act as irritants and cause the exudation of serum or an increased production of mucous.

4. Whether poisonous substances can be absorbed from the colon and produce auto-intoxication is possible, but is not certain.

THE FUNCTIONAL DISTURBANCES OF THE COLON.

The most important functional disturbances of the large intestine are :

Constipation.
Diarrhœa.
Flatulence.
Pain.

CONSTIPATION.

In a healthy adult there should be a movement from the bowels once in each twenty-four hours. The fæces should be formed, of natural consistence and color. In some individuals the regular interval is shorter, in some it is rather longer, the character of the fæces remaining normal. If the bowels cease to move regularly, and the fecal matters accumulate in the colon, the patient becomes liable to a variety of disorders.

It is hardly possible to describe all the varieties of constipation, I can only enumerate some of the ordinary forms.

1. There is a very large class of cases in which the consti-

pation is entirely due to the mental condition of the patient. They believe that if the bowels do not move every day that it is a serious matter. Every pain or abnormal sensation they have is referred to the constipation. The more laxatives, cathartics or enemata they use, the worse off they are.

There is only one cure for these patients and that is to convince them that they must leave their bowels entirely alone and let them move when they get ready.

2. There are a moderate number of cases in which there is a decided effect on the health from the insufficient action of the bowels. The prominent symptoms are: headache, vertigo, abdominal pain and loss of nutrition.

There is no one rule for the management of these patients, but many of them can be cured.

CASE I. *Chronic constipation with abdominal pain and loss of flesh and strength, cured.* Male, 39 years old, April 7, 1882.

He lived in one of the small towns of New York state and was a newspaper editor.

About four years ago he began to be troubled with constipation and occasional severe attacks of pain in the abdomen. There was no nausea or vomiting and the appetite was good.

January, 1882, he was in bed for four weeks with severe pain in the epigastrium and prostration and has been miserable ever since. In order to avoid the pain he has gradually left off one article of food after another, until for the last six weeks he has only taken milk and granum.

Now he is much emaciated, feeble and listless, the eyes sunken, the face haggard. Urine 1.020, no albumen or sugar. The ascending and transverse colon are large and plashy.

The stomach washes out clean.

April 12. He is beginning to improve.

April 17. He is stronger, there is no pain, he has one soft movement every day.

After this he improved steadily until Oct. 2, when the pain and constipation returned.

Dec. 14 he came back to New York for treatment.

From January, 1883, till 1906, he has continued to be well without medicine.

There was nothing special about the treatment. The diet was made as liberal as possible and ordinary laxatives were judiciously used.

Cases like the above are often seen; they require careful management, but can often be entirely cured.

3. In a considerable number of cases of chronic gastritis there is constipation; as the gastritis improves the constipation disappears.

4. The constipation may be due to general bad health or anæmia and the treatment has to be directed principally to the relief of these conditions.

5. The character of the food, the character and quantity of the drinking water, the absence of sufficient exercise and the mode of life, all have their effect in producing constipation.

6. There is a very curious set of cases in which the patients seem to be constipated, but really the colon contains very little fecal matter for them to get rid of.

This condition is most common in elderly ladies, but I have also seen it in young people.

CASE II. *Insufficient production of fæces, long intervals without any movement of the bowels; recovery.* Male, 17 years old, the son of well-to-do people and living under very good conditions.

Jan. 6, 1890, he had a severe attack of influenza with nausea, headache and fever.

After three weeks he was better and able to go out of doors.

Feb. 9 he had a second attack of influenza more severe than the first, with great repugnance to food, nausea, headache and sleeplessness. This lasted until March 4, when he began slowly to improve.

April 13 he was moved to the country. He improved, but the bowels were sluggish, and although he took many cathartics they only moved every four or six days.

June 14 he was moved to the seashore.

In July he could get about and play tennis. From March his temperature had been about 94°, now it got up to 98.6°.

July 28 he was caught in a squall while out sailing and had to work hard. This was followed by headache, nausea and vomiting.

From August 4 to September 27 he had no movement from the bowels, although he took many cathartics. He then passed a small quantity of hard fæces.

Then the bowels did not move again until October 10, when he passed some hard fæces. He was eating very small quantities of bread, butter, milk, eggs, meat and vegetables. He could go about, but had headaches, sleeplessness, nausea and occasional vomiting.

Nov. 5. He had a small, hard movement.

Nov. 6. He came to New York. I found him thin, pale, languid, headachy, sleepless and irritable. The intestines seemed to be empty. I thought that his bowels had not been moving, because there was so little fecal matter to come away.

He was ordered one pound of scraped beef in sandwiches, to be divided into four meals, for each 24 hours. One hour before each meal eight ounces of hot water containing 20 grains of magnesium sulphate and 10 drops of dilute sulphuric acid. He also had general massage and was to study with a tutor one hour a day. This plan was carried out and on Nov. 14 he went home, where the same system was pursued.

Nov. 24. He had a small, hard movement from the bowels.

Dec. 2. He came back to New York. He had gained two pounds in weight, there was no more vomiting, his color was better, he was stronger.

He was ordered 1½ pounds of scraped beef in three sandwiches, the fourth meal to consist of bread and honey, or baked apples and cream, the hot water was continued without the magnesium sulphate, but with the sulphuric acid. Every morning he was given an enema of 8 ounces of olive oil, he was to hold this for half an hour and then go to the water closet. He drives out in the afternoon.

Dec. 28. He has gained two pounds more in weight, the abdomen is fuller, nearly every day he passes some fæces. The beef is increased to 2 pounds.

Jan. 4, at 8 A. M., bread and milk.

At 8.30, oil enema.

At 11 A. M. the stomach is washed out.

At 1 P. M., chops and bread.

At 6 P. M., milk, cream and bread.

Jan. 13. He rides on horseback every day. He takes a pill of rhubarb and colocynth at night.

March 3. His bowels move nearly every day, he is in every way stronger and better, but any change in his diet causes vomiting. It was necessary to continue the regulation of his food and mode of life, all medicines were gradually discontinued and by July, 1893, he was a strong, healthy boy.

7. There are the every day cases of men and women who always have to take laxatives or enemata and never really get out of the habit.

RETENTION OF FÆCES.

1. A lump of hard fecal matter retained in the colon and irritating it may cause repeated and continued vomiting. In elderly persons there is, first, loss of appetite and a feeling of general depression and discomfort lasting for several days. Then they begin to vomit and continue to do so as if they had an acute gastritis. But if the abdomen is examined a hard lump of fæces can be felt in some part of the colon. If this is removed by laxatives and irrigations the vomiting stops.

2. In young and middle-aged adults constipation may be prolonged for many days, and the fecal matters accumulate in the ascending and transverse portions of the colon. When this is the case the patients often become quite seriously ill. They are in bed with severe abdominal pain, an anxious face, some distension of the abdomen, and a considerable rise of temperature. Many of them look as if they had general peritonitis. In some of them the fecal tumor can be felt. The cases vary as to the relative prominence of the fever, or the fecal tumor, as symptoms.

As a rule, by the use of calomel, castor oil, and enemata, the colon can be emptied and the patient recover.

CASE III. *Retention of fæces simulating peritonitis.* Female, 20 years old, Jan. 16, 1887.

She has always been constipated, usually having a movement at intervals of from two to seven days.

Three weeks ago she began to have pain in the lower part of the abdomen and the bowels have not moved at all during that time.

For the past week there have been nausea and occasional vomiting. Now she is well nourished, the tongue coated, temperature 103°, pulse 100, breathing 24. There is a good deal of pain and tenderness over the abdomen, but no tumor.

She was given 10 grains of calomel that night, followed by an ounce of castor oil the next morning.

Jan. 17. Three movements from the bowels, temperature 99° to 100°.

Jan. 18. The bowels continue to move, temperature 98.6°. She is given a pill of aloes and strychnine.

Feb. 10. The bowels have moved every day; she is well.

CASE IV. *Retention of fæces with symptoms like those of peritonitis; recovery.* Male, 27 years old, Feb. 25, 1879.

Before the present attack his health had been good and his bowels were moved regularly every two days.

Four days ago he was attacked with a chill, fever, headache, pain and tenderness over the abdomen. These symptoms have continued with frequent chills and no movement from the bowels.

Now the tongue is coated and dry, temperature 104°, pulse 100, breathing 34, well marked tenderness in the right iliac fossa. He was given calomel, followed by castor oil.

Feb. 26. He had a large movement from the bowels.

Feb. 27. Temperature 100° to 102°, less abdominal tenderness, tongue dry, no movement from bowels.

Feb. 28. Feels better, bowels moved.

March 1. Temperature normal, given castor oil.

March 2. Two large movements; feels better.

March 9. Has had no movement for two days, temperature 103°, was given magnesium sulphate.

March 10. He had three large movements.

After this he was well.

3. Retention of fæces simulating diarrhœa.

The rectum or the entire colon may become filled and distended with fecal matter and yet the patients have diarrhœa.

In the milder cases the condition is not constant, but occurs from time to time. The patient fails to have an operation of the bowels for several days. He feels dull, languid, loses his appetite, has headache, is troubled with flatulence and uncomfortable feelings in the abdomen, which may even amount to colic. After a few days there is a slight diarrhœa. The passages are small, painful, and do not give a feeling of relief. The patient is, at the same time, very much prostrated, vomits his food, and may even take to his bed. If you are called to attend these patients after the diarrhœa has begun, it is very important that you should recognize the true nature of the case. The administration of any preparation of opium, or of any drug which merely checks the diarrhœa, only does harm and prolongs the sufferings of the patient. A mild laxative, on the other hand, will very promptly relieve all the symptoms. The ordinary dinner pill is one of the best preparations for this purpose. Enemata of castor oil, and olive oil mixed together are serviceable.

In the more severe cases the symptoms come on gradually. The patient is at first only a little constipated; the bowels move every few days, either of themselves or with an enema, or with some laxative. And yet during this time the large intestine is not really emptied, but there is a constant accumulation of fæces in the rectum. The constipation becomes gradually more pronounced, and the patient finds that enemata and mild laxatives no longer give him a movement. Then he may use more active purgatives, which produce a number of fluid stools, and yet do not empty the large intestine of the hardened fæces, which are still accumulating. So the patient goes on from bad to worse, alternating between constipation and diarrhœa, always uncomfortable, often with very severe pain in the abdomen, losing strength rapidly. If the condition is not relieved, a person may be so reduced in this way as to die without any other disease than constipation.

The first point in the treatment is to introduce your finger into the rectum and ascertain whether or not it is filled with hardened fæces. If it is, the fæces must be scooped out with the finger or some convenient instrument, and then the rectum should be washed out repeatedly until it is entirely emptied.

After this the patient must be constantly watched and examined from time to time to ascertain that the fæces are not accumulating again. The diet must be regulated and laxatives given.

CASE V. *Retention of fæces simulating diarrhœa; death.* Male, 47 years old, Nov. 9, 1880.

Three weeks ago he began to have diarrhœa and pain in his abdomen, the diarrhœa was profuse and has continued.

Now he is thin and feeble, with pains in his abdomen, and diarrhœa.

Nov. 13. The diarrhœa stopped.

Nov. 17. The diarrhœa was profuse, the pulse feeble, the extremities cold.

Nov. 20. He died.

Autopsy.—There were some old opacities of the pia mater and the serum beneath it was increased in quantity. The lungs were emphysematous, œdematous, the bronchi congested and coated with muco-pus.

The heart, liver and stomach were normal.

The spleen was large and soft.

The kidneys were normal.

The colon for its entire length was much distended with pasty fæces. There was no obstruction anywhere. The mucous membrane of the caput coli was thickened and roughened.

The lower end of the small intestine was distended with pasty fæces. The mucous membrane was thickened and roughened.

CASE VI. *Retention of fæces simulating diarrhœa; death.* Male, 45 years old, Sept. 20, 1881.

A very stupid man, who only said that he had diarrhœa.

He was in the hospital for several days, was treated for diarrhœa, gradually failed and died.

Autopsy.—The brain was not examined.

The heart was normal.

The lungs were emphysematous.

The liver and spleen were normal.

The pelvis and calyces of the left kidney were dilated and the kidney atrophied. The right kidney was large and showed the lesions of chronic nephritis.

The entire length of the colon was dilated and filled with hard fæces. There was no obstruction. In the lower half of the colon the mucous membrane was blackened and eroded with small, superficial ulcers.

4. There are cases of retention of fæces that may readily be mistaken for typhoid fever. The patients have fever, diarrhœa, a dry tongue and cerebral symptoms.

CASE VII. *Retention of fæces with diarrhœa, abdominal pain, delirium and fever, resembling typhoid fever; recovery.* Male, 25 years old, Feb. 17, 1881.

Two weeks ago, after feeling weak and miserable for three days, he was attacked with pain in the abdomen, diarrhœa, headache, fever and delirium. The pain in the abdomen and the diarrhœa continued.

Now the tongue is brown and dry, alternating stupor and delirium, temperature 103°, pulse 116, breathing 30. In the right iliac fossa there is a hard tumor the size of a man's fist.

Feb. 18. Temperature 101° to 102°, delirium and stupor; he was given castor oil.

Feb. 19. Temperature 98° to 102°, the bowels moved well.

Feb. 21. Temperature 99°, he is still stupid. The bowels continue to be moved with castor oil.

March 1. The tumor in the right iliac fossa has entirely disappeared.

March 18. He is well.

DIARRHŒA.

A person is said to have diarrhœa if he has loose fecal or watery evacuations from the bowels instead of those of natural consistence. There is considerable variety in the composition of such abnormal stools.

(a) They may have the appearance and color of normal fæces, but contain more water, so that they are semi-fluid. Such stools may be small or large.

(b) They are of the consistence of semi-fluid fæces, but the color is white, or brown, or black. These stools may be small, or large.

(c) They are principally composed of fluid. With this fluid the fecal matter is mixed pretty smoothly, or is suspended in fragments. These stools are apt to be large.

(d) More or less mucus may be mixed with the stools.

Such changes in the composition of the stools are likely to be due to:

(a) Increased rapidity in the passage of the fecal mass from the caput coli downward, so that there is no time for the absorption of the fluid from the fæces and the formation of the natural stool in the rectum. The patients pass fæces such as they ought to be in the ascending colon, but not as they ought to be in the rectum.

(b) Changes in the functions of the glandular coat of the colon which prevent it from absorbing water. The fæces then remain for a sufficient length of time in the colon, but they are not made solid by the absorption of water from them.

(c) The character of the food and drink taken by the individual.

(d) A disturbance of the natural growth of micro-organisms in the fæces, or the addition of organisms which do not belong there.

(e) The addition of serum or mucus to the fæces.

(f) The absence of bile from the fæces.

For the treatment of the different forms of diarrhœa, therefore, we would naturally employ:

Regulation of the diet.

Drugs which affect the muscular coat and the glandular coat of the colon.

Drugs which control the growth of micro-organisms.

Drugs which control the production of bile.

Causes.—Apart from diarrhœas which are due to inflammations or new growths of the colon, we find the condition produced by a variety of causes.

Mental emotions often produce a transitory diarrhœa, which requires no treatment.

Extremes of heat and cold may cause a diarrhœa for which it is necessary to put the patient to bed, with a restricted diet and the use of a little opium.

Partly decayed fruits and vegetables, or undigested pieces of food in the intestine, often set up a diarrhœa. When this is the case it is necessary first to give a purgative to remove the irritative substances, and afterward to use opium.

Drinking-water which contains an excess of inorganic or organic substances is a frequent source of diarrhœa; some persons being especially susceptible to this source of irritation.

A good formula for the simple looseness of the bowels which is produced in so many ways is:

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Morphia Sulphate	gr. 1-30
Pulvo Cubebæ	gr. ii
Bismuth Beta-naphthol	gr. iii

Make one capsule.

A capsule is to be taken after each loose passage.

I employ the term "Morning Diarrhœa" to designate the disease of a group of patients who, either continuously or at intervals, have, during the morning hours, one or more loose passages from the bowels composed of fecal matter and of fluid.

Lesions.—As the condition is not a fatal one we have to judge of the probable condition of the colon from the symptoms. The large quantity of fluid in the discharges indicates a considerable exudation of serum from the blood-vessels of the colon. The occasional presence of mucus denotes a morbid change in the functions of the mucus-producing glands. The complete change in the character of the movements, which in some patients occurs within twenty-four hours, would seem to show that there are no structural changes in the colon, while, on the other hand, the persistence of the diarrhœa in some cases points to the existence of a productive inflammation.

Etiology.—The disease is seen in New York in a large number of persons of all classes. I have not been able to determine any satisfactory reasons for its occurrence.

The Diarrhœa.—The discharges from the bowels are of large size, or brownish color, and composed of fluid mixed with fragments of fecal matter. Their odor is often offensive. The discharges come with a rush, and are often accompanied by the escape of wind. In the more severe cases there is also from time to time a discharge of mucus. The passages are painless, or are preceded by colicky pains.

Varieties.—1. The simplest cases are those in which there is only one loose movement every morning after breakfast. This may go on continuously month after month, and year after year. Or there may be intervals of formed passages, or of constipation. There are no disturbances of the general health. Most of the patients do not seek for medical advice.

2. Much more uncomfortable is the condition of those patients who, instead of one loose movement after breakfast, have several such movements. This condition also may continue for years, or may only occur from time to time. These patients are liable to a number of additional symptoms. In some of them there is a well-marked feeling of exhaustion after the passages. The exhaustion is so great that the patients are good for nothing all the morning, and it is not until the afternoon that they are able to attend to work or business. In others the colicky pains before the passages are very troublesome. The patients get up every morning with the knowledge that after breakfast they will have to go to the water closet a number of times, and in addition will have to suffer more or less pain. Instead of the colicky pains there may be a fixed pain referred almost constantly to some one part of the colon. This pain is so severe and so constant that it may lead to the suspicion of cancer, or of appendicitis.

Mental and nervous disturbances are associated with morning diarrhœa in two entirely different ways. First, there is a nervous condition caused by the diarrhœa, and which disappears when the diarrhœa is cured. The condition is that of apprehension, of fear. The fear may be of cancer, of peritonitis, of appendicitis, or of some other definite danger, or it may only be the feeling of fear without any special object. Second, there is a nervous condition which is not caused by the diarrhœa, but which makes the diarrhœa much worse. It is an irritability belonging to the individual, of such a character that any anxiety, or trouble, or fatigue, will bring on the diarrhœa. In some patients the treatment of the diarrhœa is rendered hopeless by the frequent relapses due entirely to such a nervous condition.

3. As the disease becomes more marked the loose passages occur earlier in the morning. The patients wake up at four,

five, or six o'clock in the morning with the feeling of an impending movement of the bowels. Then they go on to have several loose movements, both before breakfast and after. The exhaustion and discomfort of the patients are very much increased by this early occurrence of the diarrhœa.

4. In some patients there is added after a time a sort of relation between the stomach and colon of such a character that, whenever food is taken into the stomach there is a movement from the bowels. This is very annoying, for the patients are obliged suddenly to leave the table during the course of every meal.

5. The occasional presence of considerable mucus in the discharges belongs to the cases which last the longest and are the hardest to cure.

Treatment.—If the disease occurs in women, before beginning any medical treatment it is important to have cured any lacerations of the perineum or the cervix, displacements of the uterus, or disease of the Fallopian tubes which may exist. The methods of treatment which are ordinarily employed are:

1. *Change of Climate.*—The effects of this are often very satisfactory and in the milder cases very prompt. A person who has a morning diarrhœa for months may leave New York in the afternoon and the next morning begin to have formed passages. Unfortunately a return to the city may be followed by a return of the diarrhœa. In the more severe cases a prolonged residence in a dry inland climate may effect a cure.

2. *Diet.*—The plans of diet usually followed are: *a*, An exclusive diet of milk; *b*, an exclusive diet of beef and hot water; *c*, a diet composed of milk and meat alone; *d*, a diet from which only the sugars and starches are excluded.

As regards the effects of treatment by diet we find: that some patients are cured; some are benefited for a time; in some there is no effect at all; some get worse.

3. In a small number of cases the diarrhœa can be cured by daily lavage of the stomach.

4. *Drugs.*—As a rule, the number of the passages can be

checked for a moderate length of time by the preparations of opium. The improvement only lasts while the opium is taken, and it is evident that the use of this drug ought not to be continued for any length of time.

The most useful drugs are castor oil, salol, resorcin and belladonna.

FLATULENCE.

This word is used to designate accumulations of air and gas in the stomach and intestines.

Of the gases in the stomach a part consists of air swallowed with the food, part of carbonic acid diffused from the wall of the stomach, partly of gases regurgitated from the intestine.

In the colon sulphuretted and carburetted hydrogen are the gases most constantly present. They are believed to be formed chiefly by changes in the fæces, but may also be diffused from the wall of the gut.

Concerning the nature and causes of the large dilatations of the colon which occur in hysteria and the infectious diseases, we need not speak at present.

We are interested in the ordinary accumulations of gas which cause feelings of pain, distension, oppression, and a great many abnormal sensations. There is nothing more common in ordinary practice than the patients who complain of a great variety of pains and abnormal sensations in any part of the body and ascribe it all to gas in the intestine.

As a matter of fact most of these patients have no more gas in their stomach and intestines than belongs to their age. If you attempt to treat the flatulence as such they are more likely to get worse than better.

If, on the other hand, the flatulence is left to itself and the attempt is made to improve the habits and general health of the patient, cures are not difficult.

PAIN.

Intestinal Colic.

This name is given to attacks of pain due to spasmodic contraction of the muscular coat of the colon.

Such attacks may be due to the presence in the intestine of irritating pieces of food, or fecal matter. In some persons the colon seems to be much more irritable than it is in others, so that attacks of colic are easily produced.

Symptoms.—The attacks are apt to come on suddenly, with pain as the first symptom, and, in the mild cases, the only symptom. The pain is referred to some part of the colon, or to any part of the abdomen. Besides the pain, there may be retraction, tenderness on pressure, distension or hardness of the abdominal wall, nausea and vomiting, a skin cold and bathed in perspiration, rapid and feeble heart-action, and a rise of temperature.

The attacks vary greatly as to their severity and duration. The mild attacks last half an hour, or a few hours, the pain is the only symptom, and it is evident that the patients are not seriously ill. The severe attacks last for days, with nausea and vomiting, a good deal of tenderness as well as pain over the colon, prostration, and sometimes a febrile movement, so that the patient may look very badly. The same patient may have one attack of colic, or several. It may be very difficult to distinguish intestinal colic from biliary colic without jaundice.

Treatment.—The object of treatment is to relax the muscular spasm and to remove irritating substances from the intestine. To relax the muscular spasm we use hypodermic injections of morphine and belladonna, hot fomentations over the abdomen, and hot-water enemata. To remove the irritating substances we give castor oil or some other purgative.

In some persons the predisposition to attacks of intestinal colic exists in an unusual degree. These persons will go on year after year with repeated attacks, at first at intervals of months, then at shorter intervals, until finally the attacks may occur every day. These patients also usually suffer from flatulence and constipation. When it takes this protracted form intestinal colic becomes a serious matter. The patients lose flesh and strength, they are unable to work, the severe pain gets them into the habit of using opium.

Of these protracted cases some are easily relieved by treatment, others are very troublesome. It may be necessary to put them for a time on an exclusive diet of scraped beef, or it may be sufficient to exclude milk, tea, coffee, beer, soup, and most of the fruits and vegetables. The bowels must be kept open and sufficient exercise insisted on as soon as the patient can bear it. Opium must only be used at the time of an acute attack; it must not be continued for any length of time. The drugs which seem to be of the most service are: belladonna, ipecac, nux vomica, cannabis indica, and sodium sulpho-carbolate.

Continuous Pain in the Colon.—There are persons who for weeks or months suffer from pain in some part of the colon, either with or without intercurrent attacks of colic.

The portions of the colon to which the pain is usually referred are the caput coli, the colon under the liver, and the colon under the stomach. The pain varies in its severity, but may be very bad and may radiate in different directions. It is a nearly continuous pain with exacerbations. No physical signs can be made out except in some cases a dilatation of the caput coli.

In making the diagnosis one has to be very careful to exclude appendicitis, cancer of the colon, calculi in the gall-bladder and cystic duct, movable kidneys, gastric pain, and aneurism of the abdominal aorta.

Treatment.—This pain is not cured by diet, nor by opium, nor by antiseptics. The treatment is empirical. The most useful drugs are combinations of nux vomica with sodium bromide, or sodium sulpho-carbolate; belladonna and cannabis indica, or small doses of castor oil, or asafœtida.

I. THE INFLAMMATIONS OF THE LARGE INTESTINES

These inflammations are commonly spoken of by the names of colitis or dysentery. The most complete account of these inflammations is the volume treating of the Alvine Fluxes in the "Medical History of the War of the Rebellion," by Dr. J. J. Woodward.

He says that it is convenient to embrace under the name of acute dysentery all the various forms of acute alvine flux, whether mild or severe, in which tenesmus is a prominent symptom. He divides dysentery into two main groups: catarrhal dysentery and diphtheritic dysentery.

He says also that the phenomena of diarrhœa and dysentery result from various degrees of irritation or inflammation of the mucous membrane of the alimentary canal. The causes of diarrhœa and dysentery are, therefore, to be found in the conditions capable of producing irritation or inflammation of this mucous membrane.

I think it may be safely said that, in the United States, most physicians mean by diarrhœa—loose bowels due to irritation of the large or small intestine; by dysentery—acute inflammations of the colon with small, frequent, painful passages of blood and mucus.

It has long been noticed that in tropical countries dysentery is especially frequent and fatal. Within a few years it has been determined that most of these cases are due to infection either with a specific amœba, or a specific bacillus.

Since 1903 a great deal of work has been done to demonstrate that the different varieties of bacillus dysenteriæ are the cause of acute endemic, epidemic, sporadic and institutional dysentery all over the world and also of the summer diarrhœa of infants. Acting on the belief that there is a disease—dysentery—due to a specific bacillus, attempts have been made to produce a curative serum by immunizing animals.

It would be very satisfactory to know that all the inflammations of the colon are due to the bacillus dysenteriæ, and that they can be satisfactorily treated by a serum. My experience is local—confined to New York and the adjacent country, with occasional imported cases of tropical dysentery.

It has seemed to me that there is no question that the tropical cases of colitis are due to infection with either bacillus dysenteriæ, or amœba dysenteriæ.

Of the ordinary forms of colitis seen in New York a certain number behave as if caused by pathogenic micro-organisms. I do not think they are all caused by the same organisms, some of them have all the characters of streptococcus infections.

In New York, while the disease is not as prevalent as it was twenty years ago, yet the variety of cases is considerable, so that in the course of years one may see a number of cases of inflammation of the colon which differ widely from each other.

In the small towns near New York there are from time to time epidemics in which a large number of people are attacked at the same time.

I. The most common of all the forms of colitis and the one with which every physician is familiar is that in which there are no structural changes in the wall of the colon, nothing but congestion, an increased production of mucus, an exudation of serum, sometimes considerable bleeding.

For practical purposes it is necessary to divide this class of cases into two varieties: one characterized by congestion, bleeding and the increased production of mucus, the other by the large exudation of serum.

1. *Colitis with Congestion and an Increased Production of Mucus.*

The inflammation begins regularly in the rectum and either remains confined to this part of the rectum, or extends upward. But in children a large part of the colon and the lower end of the ileum may be inflamed from the first. The glandular and connective tissue coats are more or less swollen, congested and infiltrated with serum and a few leucocytes. There is an increased production of mucus, which coats the inner surface of the colon and is passed by the patient. There is often a moderate bleeding from the surface of the inflamed mucous membrane. In epidemics of colitis the bleeding may be considerable. The epithelial cells which line the glandular tubules are distended with mucus, the superficial cells desquamate and are mixed with the mucus. The solitary lymph nodes in the wall of the intestine are often swollen. In children there may also be an infiltration of the glandular coat with pus cells and necrosis of the swollen lymph nodes with the formation of little ulcers.

It is the popular belief just now that some form of the dysentary bacillus is the cause of all cases of acute catarrhal colitis. The Shiga bacillus, the Flexner bacillus and some others have been frequently found in the stools. It seems to me, however, that while the epidemics of colitis are probably due to some pathogenic micro-organism, the sporadic cases may be due to a variety of irritants. In New York continued hot weather seems to be a real cause of inflammation of the skin, the stomach and the colon.

If the inflammation is confined to the lower end of the colon it regularly runs its course within a week and the patient recovers. The principal symptoms are the local ones: pain in the rectum, an irritability of its mucous membrane, which makes it try to discharge everything in contact with it, and the frequent passage of small quantities of blood and mucus. But little fecal matter is discharged. In addition there is a moderate rise of temperature, with more or less prostration. Some of the patients do not go to bed, some do not even stop work.

If, however, the inflammation not only involves the rectum, but extends up into the colon, the patients are more seriously ill, especially if they are young children. The quantity of mucus discharged from the bowels is considerable, and very little fecal matter is passed for one or two weeks. As the inflammation subsides the quantity of mucus decreases and that of fecal matter increases. In these patients the temperature is higher, the duration of the disease longer, and the prostration greater. Adults regularly recover, but children often die.

In epidemics the quantity of blood discharged from the bowel may be considerable, there is a small number of severe and tropical cases of colitis are due to infection with either bacillus dysenteriæ, or amœba dysenteriæ.

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In epidemics the quantity of blood discharged from the bowel may be considerable, there is a small number of severe and even fatal cases, but they run their course within one or two weeks.

CASE VIII. *Acute catarrhal colitis.* Female, 8 years old.

Tuesday afternoon. Temperature 102°, nausea, pain and tenderness over the appendix.

Tuesday night. Temperature 104°, a good deal of abdominal pain, eighteen passages of serum and mucus.

Wednesday morning. Temperature 100°, no pain, ten passages containing blood and mucus in the twelve hours.

Thursday morning. Temperature 99°, no pain, feels well, three passages conaining mucus in the night, eight passages in the day.

Friday morning. No passage in the night.

Saturday morning. She has had formed movements, containing some mucus in the twenty-four hours, has no pain, is hungry and feels well. After that she was well.

The treatment was rest in bed, a milk diet and small doses of beta-naphthol bismuth with codeia.

CASE IX. *Acute catarrhal colitis.* Male, 35 years old, April 26.

In the morning at 3 o'clock he began to have small loose movements, composed principally of blood with a little mucus. There were seven passages with abdominal pain and tenesmus, moderate prostration.

Temperature 98°, pulse 62, colon full of gas.

He was put to bed, given hot rectal irrigations and small doses of morphine, cubebs and bismuth.

April 27. He had a formed movement, and after that was well.

The treatment of these patients is usually satisfactory. They must be kept in bed on a diet of milk or gruel until the colitis has entirely subsided. Small doses of opium with one of the preparations of bismuth and irrigation of the rectum with infusions of flaxseed are good treatment. If there is any retention of fecal matter in the colon castor oil or sulphate of magnesia are the best laxatives.

2. *Acute colitis with a large exudation of serum.*

These cases are not to be confounded with the cases which I have described elsewhere under the name of "morning diarrhœa." The condition is an inflammatory one, and not a functional disturbance of the intestine; they differ also from the cases of ptomaine poisoning.

The disease is not a fatal one, and I have never had the opportunity of seeing the colon of one of these patients. The character of the symptoms is such as to make one believe that the inflammation involves the upper part of the colon, and that the exudation of serum into the cavity of the gut is the principal feature of the morbid process.

This form of colitis is especially common when, during the summer, there is a week of continuous hot weather. Cases are seen, however, at all times of the year. How far water or milk or food may be responsible for an attack it is difficult to say. In some persons there is a well-marked predisposition to this form of colitis, so that they suffer from attacks summer after summer.

The patients have at first a feeling of discomfort in the abdomen or colicky pains, a moderate amount of prostration, and sometimes nausea. Then there is the feeling of the necessity for emptying the bowels. The patients go to the water closet, sit down, the sphincter opens, and a large quantity of fluid runs out without any effort of expulsion. The fluid consists of fluid fæces, serum, and mucus. The quantity of each discharge is considerable, sometimes as much as two quarts. There may also be nausea and vomiting.

There are a number of such discharges during each twenty-four hours. The patients are not always confined to bed, but they are weak and miserable. As a rule, there is little febrile movement, but the temperature may run from 101° to 103°. Such an attack may only last for a few days, or it may continue for weeks or months, the gastritis also may continue.

Treatment.—A large number of the cases get well under a variety of treatments, or even without any medication at all; but, on the other hand, there is a considerable number of cases in which the disease continues in spite of treatment. Every year I see several patients in whom the colitis has continued for months in spite of treatment by diet, opium, bismuth, and the intestinal antiseptics.

And yet all of these cases can be cured if sufficient pains is taken. The diet and mode of life must be regulated to suit each patient. The old drugs—opium, castor oil, belladonna, bismuth, salol, resorcin, etc., answer the purpose.

The point of importance is to select the drugs or combination of drugs suitable to each case.

CASE X. *Acute catarrhal colitis with serum.* Male, 35 years old, Dec. 4.

Yesterday, without any indiscretion in food or drink, he was taken with pain in the abdomen, chilly feelings, vomiting, and had fourteen large, serous passages in the twenty-four hours; temperature 99°.

He was put to bed and given codeia, salol and castor oil in small doses.

Dec. 5. He was better.

Dec. 6. He was well.

CASE XI. *Acute catarrhal colitis with serum.* Male, 43 years old, July 5.

Nine days ago, after suffering from the heat and drinking a good deal of ice water, he began to have nausea and vomiting, cramps in the abdomen and profuse watery diarrhœa, fifteen to twenty stools a day.

Now, temperature 101°, pulse 108, breathing 30. He feels weak and miserable, with the profuse, watery diarrhœa continuing.

The next day he left the hospital unimproved.

II. CHRONIC CATARRHAL COLITIS.

The acute catarrhal inflammations of the colon may be followed by chronic ones, or these may be of subacute or chronic type from the outset. Chronic catarrhal inflammation follows one of two types:

1. The cases in which the only anatomical change seems to be an increased production of mucus. Whether this is an inflammatory condition, or a disturbance of function of the glands of the colon I do not know. It is of very ordinary occurrence in New York, especially in women.

It is seen in all grades of severity. The mucus is passed by itself, or mixed with the fæces. The quantity of mucus is small or considerable. It is passed before, with or after the fæces, according to the position of the part of the colon that is diseased. The fæces themselves are formed, or fluid.

In the simplest cases there are no symptoms. The patients at some time discover they are passing mucus, that is all.

Next in order come the patients in whom the only symptoms are abdominal pains and loss of nutrition.

Then come the large number of cases with all sorts of disturbances of the functions of the stomach, intestines and liver, loss of nutrition and an endless variety of nervous phenomena.

The treatment is sometimes satisfactory, sometimes very much the reverse. The general management and diet count for a great deal. Rest in bed, regulated exercise, change of climate and mode of life according to the condition of the patient are to be attended to.

Rectal irrigations may be of very great service.

Of the drugs: resorcin, castor oil, belladonna, ipecac, cubebs, bismuth are some of the best.

Case XII. *Chronic colitis with mucus.* Male, physician, 40 years old.

For one year he has had several loose fecal movements, with a discharge of mucus following the fæces, every day. This condition, in spite of treatment, has continued without improvement. He has lost a good deal of strength and some flesh. He was given salol with castor oil, and bismuth, rhubarb and ipecac on alternate days.

After two weeks there was moderate improvement.

The castor oil with salol was replaced by resorcin and sulphocarbolate of soda. After this he improved steadily until after three weeks he had regularly one formed fecal passage, without mucus, every day. He also felt well and strong.

The cure was permanent.

CASE XIII. *Alternating constipation and diarrhœa, with large discharges of mucus.* Female, 30 years old, Oct. 1, 1902.

She has always been constipated. Two years ago the constipation began to alternate with periods of diarrhœa, and she began to pass considerable quantities of mucus. The mucus was in strings, it followed the movements or was passed by itself. The attacks of diarrhœa were accompanied with abdominal pain, flatulence and nausea. They have become gradually worse, the last one kept her in bed for two weeks.

She has been variously treated without improvement.

She was given resorcin and sulpho-carbolate of soda.

Nov. 1. She is much better, no more attacks of diarrhœa and less mucus.

December 30. She feels well, no diarrhœa, no mucus.

She was still well a year later.

There seems to be no very sharp dividing line between the bad cases of this kind of colitis and the cases of so-called membranous enteritis.

CASE XIV. *Chronic colitis with mucus.* Male, 26 years old.

In December, 1905, began to have general abdominal pain of varying severity, but occurring every day, the abdomen was somewhat distended, he had formed movements at irregular intervals.

Two weeks later he passed a piece of mucus about two inches square. After that he passed considerable quantities of mucus every day. The mucus was passed before the stools or by itself. He continued to have more or less abdominal pain.

He was treated in a variety of ways, but without improvement.

March 18, 1906, he came to me.

He was put on castor oil and salol by the mouth, rectal irrigations with chloride of zinc and a liberal diet.

March 25. There was no more pain or mucus. He felt very well and went home with instructions to continue the salol and castor oil and irrigate the rectum every other day.

April 26. He came back with some return of the mucus, but no pain. He was ordered to continue the irrigations and to alternate the salol and oil with small doses of belladonna, ipecac and rhubarb. He improved so that by the end of May he gave up the irrigations.

Since then there has been no pain or mucus and he has been very well up to the present time, Oceober 16, 1906.

MEMBRANOUS ENTERITIS.

This is one of the most curious and perplexing affections of the colon. It seems to be a form of inflammation and yet does not behave like other inflammations. It is of not uncommon occurrence, but autopsies are rare and when we get them they tell us but little.

It occurs much more frequently in women than in men, and is a disease of adult life. It is especially common in women who have disease of the uterus, the ovaries, or the Fallopian tubes.

The striking feature of the disease is that the patients discharge from the bowels shreds, ribbons, or tubes composed of fibrin and mucus. In all the other forms of colitis mucus is discharged by itself, but in this form the discharges are rendered quite different by the mixture of fibrin with the mucus.

There is considerable variety as to the persistence and the severity of the symptoms.

1. There are patients who suffer from gastric disturbances, abdominal pain, loss of nutrition and a variety of nervous phenomena. If they are women, they often have disease of the uterus, ovaries, or tubes. The bowels are sometimes constipated, sometimes loose. From time to time they pass shreds, ribbons, or tubes composed of mucus and fibrin. They may continue in this way for months and years and then recover.

2. There are patients in whom the disease manifests itself by attacks which occur at intervals. Between the attacks their health is fairly good. From time to time they suffer for one or more days from abdominal pain, large diarrhœal passages, and the discharge of large quantities of mucus and fibrin. These patients also often recover.

3. There are the bad cases in which the disease continues. The stomach is always irritable, with nausea and vomiting. The bowels are never natural, but either constipated or loose. The mucus and fibrin are discharged in large quantities every day. These patients become extremely feeble and emaciated and may die from the disease.

Treatment.—In the ordinary cases we regulate the diet and mode of life, employ such treatment as will relieve the gastric dyspepsia, the constipation, or the disease of the uterus, ovaries, or tubes. For the condition of the bowels we employ irrigations, castor oil, ipecac, and other drugs.

In the bad cases good results are reported from opening the caput coli and keeping open an artificial anus for some months.

2. *Chronic catarrhal colitis with changes in the wall of the colon.*

The inflammation is confined to the rectum, or extends upward and involves a considerable part of the colon. There is constantly an increased production of mucus. The glandular coat alone, or all the coats of the colon are thickened by a growth of new connective tissue. There may also be little polypoid outgrowths composed of connective tissue and hypertrophied tubules. It is found, however, in some of the fatal cases that the changes in the wall of the colon are not as profound as one would have expected from the symptoms.

The patients go on for months or years with frequent small passages of blood and mucus and more or less discharge of loose fecal matter. Unless the inflammation is confined to the rectum they seldom recover, although they often improve for a time. There is a progressive loss of flesh and strength.

The treatment is apt to be unsatisfactory. Not much is accomplished by the regulation of the diet or the use of drugs. Irrigations do something. Prolonged residence in a dry inland climate is sometimes of great benefit.

CASE XV. *Chronic catarrhal colitis, death.* Male, 46 years old, Jan. 3, 1890.

Seven years ago he began to have diarrhœa, sometimes with blood in the stools. The diarrhœa has continued steadily through all this time and he has gradually lost flesh and strength. He is now emaciated and feeble, temperature 99°, urine normal, twelve loose passages daily.

He continued to get steadily weaker, and died on Feb. 6.

Autopsy. The heart, lungs, liver, spleen and small intestine were normal.

The colon was somewhat thickened for its entire length, but more so in the rectum. There was an extensive growth of small celled connective tissue, which separated the tubules of the glandular coat and in some places had entirely obliterated them.

CASE XVI. *Chronic catarrhal colitis.* Male, 37 years old, March 25, 1882.

In 1864 he had an attack of dysentery which lasted for three months. From this he recovered entirely and remained well until August, 1881. Then he began to have, first, an unnatural appetite, then pain in the epigastrium, then diarrhœa.

After three weeks he began to pass mucus and after three months blood. There were from nine to twenty small fluid passages a day without tenesmus. Temperature 98.7°.

The diarrhœa continued unchecked, he lost flesh and strength, and died April 2.

Autopsy. The brain was not examined.

The heart was small.

The upper part of the lower lobe of the left lung was hepatized.

The liver, spleen and kidneys were normal.

The stomach showed the changes of chronic gastritis.

The small intestine was normal.

For the entire length of the colon its mucous membrane was thickened with superficial ulcers and small polypoid thickenings.

III. PRODUCTIVE AND NECROTIC COLITIS.

Synonyms.—Catarrhal colitis, dysentery.

The anatomical characters of this form of colitis are: an increased production of mucus, an exudation of serum, an emigration of white blood-cells, a growth of new connective tissue in the glandular coat and the connective-tissue coat, and small areas of necrosis of the glandular coat.

The inflammation prefers the upper part of the colon, but it may involve its entire length or be confined to its lower third.

The gross appearance of the colon is that of congestion of the mucous membrane, with perhaps a coating of mucus—the ordinary appearance of an acute catarrhal inflammation; but if we look more closely we see that in addition there are numerous small, superficial ulcers, many of them so small that they cannot be seen with the naked eye. In the old and severe cases, however, instead of these small ulcers we find a destruction of large areas of the glandular coat.

The morbid changes belong to the glandular coat, but may extend to the superficial layers of the connective-tissue coat. The destruction of the glandular coat is effected by simple necrosis or by an infiltration with pus-cells. The ulcers do not have overhanging edges. Between the ulcers some of the tubules show necrosis of their epithelium, while some are obliterated by the growth of cells between them. The tissue between the tubules is thickened by a growth of connective-tissue cells, or by an infiltration with pus-cells, or by both. There may also be a moderate infiltration of the superficial layers of the connective-tissue coat with pus-cells and connective-tissue cells.

I have never been able to make out the causation of this form of colitis. It is not infrequent in New York. It is seen both in hospital and private practice. It occurs at all seasons of the year. Whether it is caused by impure water or milk or food I do not know. The fæces show the usual variety of micro-organisms, but the general features of the disease are those of a streptococcus infection.

The invasion of the disease is sudden or gradual. If it is sudden, the patients are attacked with vomiting, frequent passages, abdominal pains, prostration, and a rise of temperature. The first passages are diarrhœal—large and small and composed of fecal matter, serum, and mucus; or dysenteric—small, painful, and composed of mucus and blood. The dysenteric passages do not continue; they belong to the first days of the disease. The prostration is considerable; the patients look sicker than they do with an ordinary catarrhal colitis. Sometimes there is even a sort of shock given by the invasion of the disease; the patients may have attacks of syncope. The regular duration of the acute cases is three weeks.

If the invasion is gradual, the patients begin with what seems to be an ordinary diarrhœa—three to five loose fecal movements a day, with more or less colicky pain or constant abdominal pain. There is at first no rise of temperature. The patients gradually lose flesh and strength, but it may be several weeks before they go to bed. And yet it is a matter of much importance that the character of the colitis should be recognized and the patients put to bed during this early stage of the disease.

As the disease goes on the passages continue to be frequent and of considerable size. They are composed of fluid fæces, serum, mucus, and often blood. The mucus is present in considerable quantities; it is diffuse or in the form of nodules. If the patients improve, the fecal matter increases in quantity and becomes more consistent, while the quantity of serum, mucus, and blood diminishes. The abdominal pain, whether colicky or constant, continues. There is no appetite. In some cases nausea and vomiting are very troublesome symptoms. There is usually a rise of temperature in the afternoon, even in the chronic cases. The patients steadily lose flesh and strength. Of the cases which recover some run their course within four weeks and recover entirely, and yet while the disease is going on the patients are sick enough to cause much anxiety.

In the fatal cases I have seen the disease prove fatal in seven days, but that was in a person already exhausted by privation and exposure. The rule is that the disease continues for a number of months before the patients die. Occasionally we see such a colitis in its early stages in persons who die from pneumonia. The prognosis is serious. A moderate number die from the disease, and in a very considerable number the disease is protracted for months.

The most important time for the treatment of this form of colitis is during the early stages of the disease. It is in the patients who are neglected at first that we see the protracted forms of the disease.

No matter how mild the invasion of the disease is, the patients should at once be put to bed and on a diet composed principally of milk, and this management must be kept up until all evidences of acute inflammation have disappeared.

In the chronic cases, on the other hand, it is important that the patients should get out of doors and eat a sufficient quantity and variety of food. As a rule, these patients do best in a dry, elevated, inland climate.

During the earlier stages of the disease irrigations of the rectum are of service, and even in the chronic cases such irrigations may be used with advantage from time to time.

As regards medication, it has seemed to me that I have obtained the best results, both in the acute and chronic cases, by the use of combinations of opium, salol, and castor oil; but I must admit that these remedies sometimes fail, and that I see patients recover under other plans of treatment.

CASE XVII. *Productive and necrotic colitis, early stage; death from lobar pneumonia.* Male, 19 years old, Jan. 17, 1895.

Eleven days ago a chill followed by vomiting and diarrhœa. Three days later he began to cough with muco-purulent sputum and pain in the left chest. He continued to have from four to seven stools daily.

Now, temperature 105°, pulse 98, breathing 28, subcrepitant and coarse rales over both lungs, over the left lower lobe dulness and bronchial breathing. He became steadily worse, temperature high, pulse more rapid, delirium, frequent yellow, watery stools without blood or mucus.

Jan. 25 he died.

Autopsy. Only the colon was examined.

The rectum contained yellow, pasty fæces, the rest of the colon was empty.

The lower third of the colon was congested and studded with very minute ulcers. These are formed by necrosis of the glandular coat. Between them the glandular coat is infiltrated with cells.

CASE XVIII. *Productive and necrotic colitis, terminal pneumonia.* Male, 54 years old, Sept. 26, 1884.

One month ago he began to have cramps in the abdomen and, after two days, from eighteen to twenty stools daily; some of the stools contained blood and mucus. He was treated without any improvement.

Now he has from twelve to twenty loose, watery and fecal passages daily, pain in the abdomen; temperature 97.8°.

Oct. 1. He had two formed stools. In the afternoon the temperature rose to 103°, he became delirious, with the physical signs of consolidation of the lower lobe of the right lung.

He died the same evening.

Autopsy. The brain was not examined.

The lower lobe of the right lung was in the condition of red hepatization.

The liver, spleen and stomach were normal.

The kidneys showed the lesions of chronic nephritis.

For the entire length of the colon its wall is soft and rotten. The mucous membrane is thickly studded with small and large, flat superficial ulcers involving only the glandular coat. The remaining glandular coat and the connective tissue coat are infiltrated with round cells.

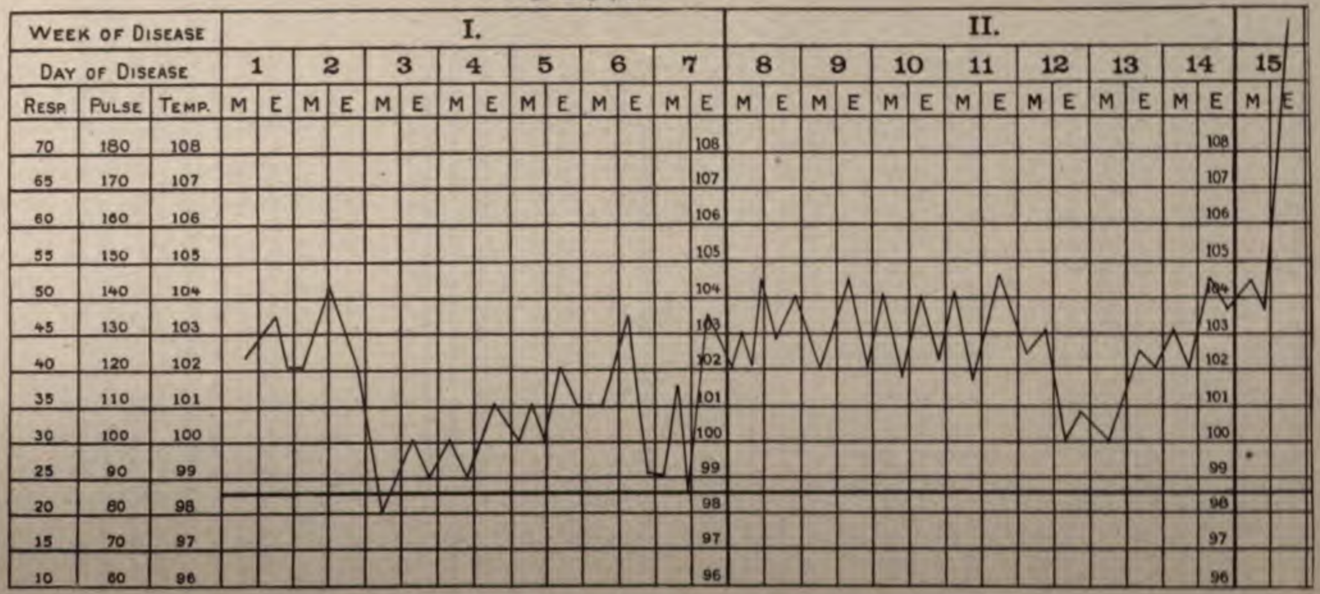
CASE XIX. *Purulent and necrotic colitis.* Male, 34 years old, June 28, 1879.

A large, fine looking man, who said his health had always been good.

Two weeks ago he began to have three to five loose fecal passages a day, without blood or mucus. There was occasional vomiting and pain in the upper part of the abdomen.

Now he has pain in the hypogastrium, three to five loose fecal passages daily, constant nausea, occasional vomiting; temperature 103°, pulse 118, breathing 30, urine 1.026, no albumen,

FIG. 1.



CASE XX.—LARGE INTESTINE. PRODUCTIVE AND NECROTIC COLITIS.

no physical signs over the heart or lungs. He is feeble, but not emaciated.

He continued in the same condition, three to five loose fecal passages without blood or mucus daily, constant nausea and vomiting; temperature 99° to 103°, gradual emaciation.

Aug. 5. There were several large, bloody passages.

Aug. 13. He was found dead in bed.

Autopsy. The body was emaciated.

The brain was not examined.

The lungs, heart and liver were normal.

The spleen was small and soft.

The kidneys were a little increased in size, the cortex pale, the surface smooth.

The lower part of the small intestine was congested, the solitary and agminated glands were a little swollen.

The colon for 18 inches above the anus was normal. Above this the glandular coat had almost entirely disappeared, leaving the muscular coat bare with little patches of mucous membrane still adhering.

Where the mucous membrane was left it was densely infiltrated with pus cells. The whole wall of the gut was split up and infiltrated with pus cells and small, polygonal cells.

CASE XX. Figure 1. *Productive and necrotic colitis; death.* Female, 48 years old, July 18, 1900.

She came into the hospital with the ordinary symptoms of emphysema and increased arterial tension.

She was discharged on July 22.

Aug. 1, 1900, she came back to the hospital with temperature 102°, pulse 120, breathing 36, nauseated, six loose brownish passages from the bowels mixed with mucus and blood.

Aug. 2. Nauseated, skin hot and dry, tongue coated, colicky pains, watery stools with blood and mucus.

Aug. 6. The nausea has continued, with two to four passages of thin fluid and blood.

Aug. 11. She has continued to have from four to six brown, watery stools, with very little blood. She is getting steadily weaker and is now delirious. She continued to have the same passages from the bowels and died Aug. 14.

Autopsy. The lungs were emphysematous.

The heart was normal, except for a little thickening of the coronary arteries.

The liver was cirrhotic.

The whole length of the colon was studded with small, round, clean-cut ulcers.

The kidneys showed the lesions of chronic nephritis.

CASE XXI. Figure 2. *Productive and necrotic colitis; recovery.* Girl, 9 years old, living in West Seventy-second street.

In September her mother had an ordinary attack of catarrhal colitis lasting for a week.

On October 25 the girl felt chilly and her temperature went up to 104°; that night the bowels were loose, with colicky pains.

Oct. 26. The temperature chart begins. She had each day four small, loose fecal passages.

Oct. 29. I saw her for the first time, the abdomen was distended with gas. She was treated with hot rectal irrigation and a combination of morphine, acetanilid and cubebs.

Nov. 1. She had ten small passages of fecal matter and mucus, the tongue was dry, she looked seriously ill. She was given small doses of salol and castor oil.

Nov. 3. She had sixteen small passages of fecal matter and mucus.

Nov. 5. The stools were involuntary, she was delirious.

Nov. 6. She began to cough, there were five stools in the twenty-four hours.

Nov. 8. There were the physical signs of consolidation of the lower lobe of the right lung; only two stools.

Nov. 11. The pneumonia is resolving, only one loose stool.

Nov. 14. She had her first formed stool without mucus.

Nov. 16. One formed stool, all medicine was stopped.

Nov. 19. She was given solid food.

Nov. 28. She began to sit up. Her convalescence was uninterrupted.

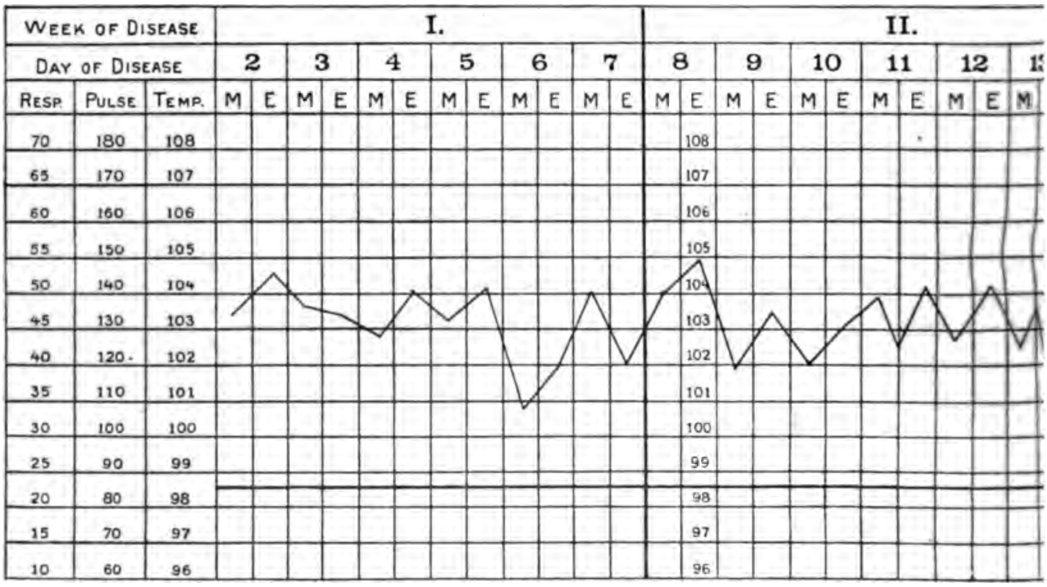
CASE XXII. *Productive and necrotic colitis of seven months duration, death.* Male, 35 years old, Dec. 9, 1885.

Six months ago he had an attack of rheumatism, from which he recovered.

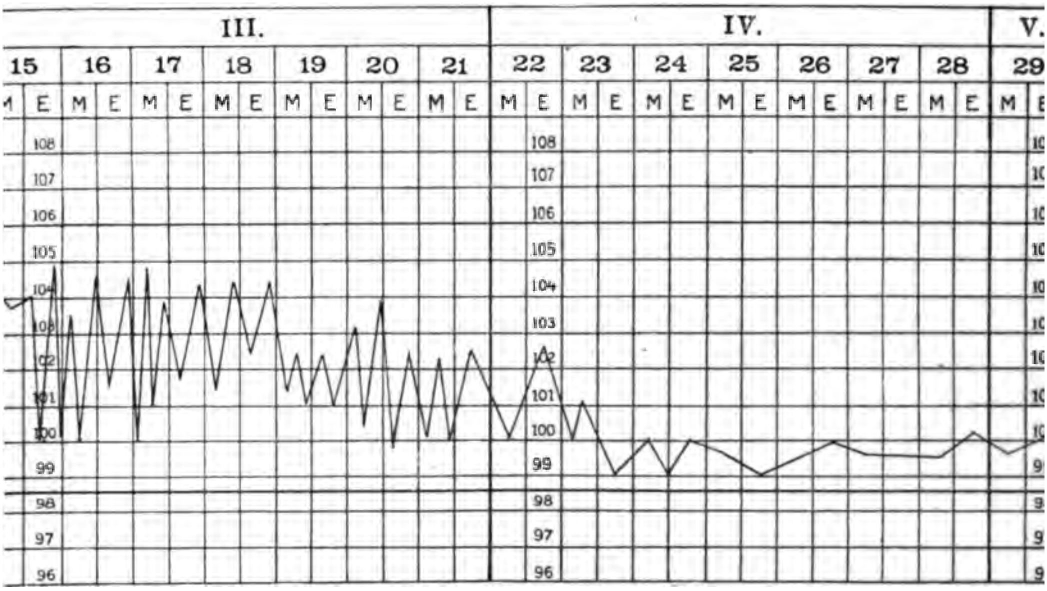
Five months ago he began to have pains in the abdomen and loose stools, with tenesmus and blood.

Now he is pale and emaciated, œdema of feet and legs; temperature 98°, frequent stools of fluid fæces and mucus.

Dec. 15. He continues to have nine to fifteen stools daily,



Case XXI. Large Intestine.



ND NECROTIC COLITIS. RECOVERY.

he also has a cough with bloody sputum, pain in the left chest, dulness and sub-crepitant rales over the lower lobe of the left lung.

He continued to have frequent loose stools, to cough, to lose flesh and strength, and died on Feb. 5, 1886.

Autopsy. The brain was not examined.

Both lungs were emphysematous, the bronchi were congested and contained muco-pus. In the lower lobe of the left lung there was a considerable area of broncho-pneumonia.

The heart was normal.

The liver was small and covered with old adhesions, but otherwise normal.

The spleen was large and firm.

The kidneys looked normal.

The colon to the naked eye showed but little evidence of disease. The surface of the mucous membrane was not coated with mucus or fibrin, no ulcers could be seen, the wall of the gut was not appreciably thickened.

Minute examination showed a very considerable growth of cells between the tubules of the glandular coat. In many places there were small areas of necrosis forming minute, superficial ulcers. The lymphatic glands were unchanged.

IV. CROUPOUS COLITIS.

There is a form of colitis characterized by the combination of an exudation from the blood-vessels, which forms fibrin, with necrosis. This is commonly called croupous or diphtheritic dysentery.

The inflammation regularly begins in the rectum. It may remain confined to this part of the colon, or it may extend upward and involve the whole length of the colon and even the lower part of the small intestine.

The irritants which cause the colitis seem to act, according to their virulence, upon the glandular coat alone, or upon the connective-tissue coat also, or upon the whole thickness of the wall of the colon. In the majority of cases the inflammatory changes are produced not uniformly, but in patches.

In the milder cases, therefore, we find the inner surface of the colon studded here and there with little patches of false membrane. Vertical sections of the wall of the colon at these points show a layer of fibrin and pus, necrosis of the superficial portions of the glandular coat, and a little infiltration of the glandular coat with fibrin and pus.

In the more severe cases the layer of fibrin on the inner surface of the colon is thicker, large infiltrations of fibrin are found in the connective-tissue coat, and even the muscular and peritoneal coats are involved in the exudation. The necrosis involves the whole thickness of the wall of the colon.

The dead tissue soon sloughs away and leaves ulcers of varying number, size and depth.

In New York, this form of colitis belongs to the autumn months. Not infrequently people bring it back to town with them from the country. In such cases there may be a history of contaminated drinking-water. The behavior of the inflammation is that of one caused by the presence of pathogenic bacteria, and such bacteria can be demonstrated in the fæces and in the wall of the colon. But it is not to be forgotten that identical anatomical changes are produced by poisoning with corrosive sublimate, so that it is possible that such a colitis may be produced by other irritants than bacteritic toxins.

Twenty years ago croupous colitis was one of the regular autumn diseases of the country about New York. Often the same persons would have the disease every autumn. It was one of the ordinary causes of death in old persons. As more care has been taken about the contamination of drinking-water and the disposal of fecal matter the disease has become less and less common.

Symptoms.—The patients have numerous small, painful passages of blood and mucus. If the inflammation extends above the rectum, they also pass large quantities of brownish fluid. There is nearly constant rectal pain, irritability, and tenesmus. There may also be colicky pains and strangury. Many of the patients vomit, especially at the beginning of the disease. There is regularly a well-marked febrile movement throughout the disease, but in some of the bad cases the temperature is low throughout. In the severe cases the prostration is marked, the heart's action is rapid and feeble, delirium and stupor are developed. The disease lasts from one to six weeks.

Prognosis.—The prognosis is serious on account of the severe character of the lesion, the general poisoning of the patient and the permanent changes left in the wall of the intestine. If the whole length of the colon is involved, the patients rarely get well. If the ulcers are large and deep, although the patient recovers from the inflammation, he is left with a colon so badly damaged that he is always an invalid.

All the patients are to be put to bed and kept on a fluid diet. Most of them require some preparation of opium to keep them comfortable. Many of them need alcoholic stimulants. It is important to distinguish whether the inflammation is confined to the rectum or whether it extends higher up. If it is confined to the rectum, it can be treated by irrigation alone; if it involves a larger part of the colon, drugs must be given in addition to the irrigation. Two quarts of an aqueous solution of corrosive sublimate, 1 to 10,000, has given me the best results as an irrigation. Ipecac, in 20-grain doses once or twice a day, seems to be the most efficient drug. I have also seen good results, however, from the early use of salol in 5-grain doses every hour.

CASE XXIII. *Croupous colitis of short duration; death.* Male, 53 years old, Oct. 26, 1884.

Four days ago he was attacked with vomiting, diarrhœa, with green and mucus stools, and severe rectal pain.

Now he is emaciated, temperature 98° to 103°, ten to twenty small, mucous stools daily.

Oct. 29. Temperature 99° to 100°.

After this the temperature was below 98°. The same small, frequent, mucous stools continued. He grew weaker, and died November 3.

Autopsy. The brain was not examined.

The heart was normal.

The lungs were congested and œdematous.

The stomach, liver, spleen and kidneys were normal.

For its entire length the mucous membrane of the colon was swollen, congested and coated with fibrin.

CASE XXIV. *Croupous colitis with secondary pleurisy and pericarditis.* Male, 37 years old, Sept. 10, 1882.

Two weeks ago he began to feel sick, with vomiting and frequent, watery stools.

One week ago he began to have frequent, small, painful passages of blood and mucus.

Now he is feeble and emaciated; temperature 99°, frequent passages of mucus and greenish fluid.

Sept. 16. The passages are less numerous, but the man is no better.

Sept. 19. He is worse, with dulness, bronchial voice and breathing over the lower lobe of the left lung.

Sept. 20 he died.

Autopsy. The brain was not examined.

The pulmonary pleura of the left lung was thickly coated with purulent fibrin. The lower lobe was in the condition of commencing red hepatization.

There was a little recent fibrin on the pericardium.

The liver was large, firm and congested.

The stomach and spleen were normal.

The kidneys were a little swollen.

About 12 inches of the lower end of the small intestine was congested and coated with fibrin.

The lower two-thirds of the colon was uniformly coated on the inside with a thick layer of fibrin.

The mesenteric glands were swollen.

CASE XXV. *Severe croupous colitis; recovery.* Male, 51 years old.

Aug. 26. He was taken ill with frequent painful, bloody passages from the bowels.

Sept. 8. Six passages of blood and mucus, pain and tenderness over the abdomen, tongue dry and brown, temperature 101°. He was ordered a starch and laudanum enema after each movement.

Sept. 9. Eight passages, temperature 100°; he was ordered an ounce and a half of castor oil.

Sept. 10. Six passages, temperature 100° to 100.5°; he was given 30 grains of ipecac and vomited three hours later.

Sept. 11. Six passages, temperature 101°, pulse feeble.

Sept. 12. Six passages, no more blood, tongue dry, temperature 100.5°.

Sept. 13. Seven passages; he was again given castor oil and ipecac; this time he did not vomit.

Sept. 14. Five passages containing more fecal matter; temperature 100°.

Sept. 15. He was again given oil and ipecac and did not vomit.

Sept. 16. Four passages, temperature 101° to 102°.

Sept. 17. Four passages, the enemata of starch and laudanum were stopped. He was given dilute hydrochloric acid and laudanum every three hours.

Sept. 18. Three passages, temperature 101°.

Sept. 22. Four passages, temperature 103°. He was given muriate of quinine.

Sept. 24. Three passages, temperature 102°.

Sept. 26. Four passages, temperature 103°. He now has some appetite for food.

Sept. 29. Three passages, temperature 102°; the hydrochloric acid and opium was stopped and he was given 15 grains of quinine three times a day.

Sept. 30 to Oct. 12. Three to four passages a day, without blood or mucus, temperature 100° to 101°. He is steadily improving.

Oct. 13 to Oct. 24. Two to three passages a day, temperature 99° to 100°. He is well enough to sit up.

Nov. 5. One normal passage a day, no more fever. He is well.

CASE XXV (*b*). *Severe croupous colitis; recovery.* Male,

Sept. 7. He had a diarrhœa that lasted for a week and then stopped.

Sept. 15. He began to have pain across the abdomen and small passages of serum and blood.

Now, temperature 99°, tongue coated and dry; small, bloody, painful passages every hour. He was given castor oil, opium and ergot.

Sept. 21. Temperature 99° to 100°; the small bloody passages continue unchecked; tongue dry. He looks very ill. He was ordered 20 grains of ipecac and a hypodermic of morphine every day.

glandular coat is undermined, and ulcers with overhanging edges are found of various sizes and depth.

(c) Large portions of the whole thickness of the wall of the colon are swollen and completely necrotic. The patients usually do not live long enough for these dead portions to slough away.

(d) The ulcers may be small and not deep, but there is a large growth of connective tissue between the tubules and in the connective-tissue coat.

The inflammation may extend from the colon to the lower end of the ileum.

There may be a general peritonitis, either with or without perforation. There may be amœbic abscesses in the liver, or in the lower lobe of the right lung. It may very well happen that the symptoms of the abscess of the liver, or those of the abscess of the lung are more prominent than those of the colitis.

Symptoms.—We find that there are acute cases which run a mild course; acute cases which run a severe course; sub-acute cases which are either mild or severe; and chronic cases.

In the mild cases, whether acute, sub-acute, or chronic, the necrosis is not large and the length of colon involved is not great In the severe cases, on the contrary, there is much necrosis and a considerable part of the length of the colon is involved.

1. *The Acute Mild Cases.*

There are frequent passages composed of fecal matter, mucus and blood. The fecal matter is pasty and mixed with mucus or the mucus is in the form of little nodules. The amœbæ are most easily found in the nodules of mucus. The patients look seriously ill, but the temperature is irregular and not at any time very high. Nausea, vomiting and colicky pains are often present. The disease runs for about three weeks and the patients regularly recover.

Treatment.—The patients should have regularly every day a large irrigation of hot salt and water. By the mouth they take either 10 minims of castor oil with 10 minims of tincture of opium and 1 ounce of brandy; or one-thirtieth of a grain of morphine with 3 grains of cubebs and 2 grains of subnitrate of bismuth three or four times a day.

CASE XXVIII. *Acute amœbic colitis; recovery.* Male, 30 years old.

Was with the army at Santiago; while there he was not sick. He was on the transport coming to Montauk from August 22 to 28. While on the transport he began to have frequent, large, loose fecal stools, without blood.

From August 28 to September 11 he was in the hospital at Montauk.

His temperature ran between 98° and 100°.

He had frequent large and small, loose fecal movements, and passed a good deal of blood. Active amœbæ could always be found in the stools.

He was treated with rectal irrigations of salt and water, and small doses of morphine, cubebs and bismuth.

By September 11 he was well.

2. *The Severe Acute Cases.*

In these cases the lesion involves the greater part of the length of the colon, the necrosis is extensive, there may be abscesses in the liver, or in the lung.

The patients are very seriously ill and look badly. They seem to be sick rather from the extensive changes in the colon than from an infection. There seems no reason to believe that the amœbæ produce a toxin.

The temperature is very irregular, high or low. Nausea, vomiting and colicky pains are often present. The patients rapidly lose flesh and strength.

The passages are composed of soft, pasty fecal matter, mixed with mucus, or the mucus is in nodules; there may also be blood. The passages are frequent, sometimes involuntary, and the quantity of fecal matter discharged is very large. The disease may cause death in a few days, or continue for several weeks, or become chronic.

The accompanying abscesses of the liver and the lung add to the symptoms and dangers of the colitis.

The prognosis is unfavorable, even if the patients seem to recover, the gut is left badly damaged.

The treatment is the same as for the mild cases, but is unsatisfactory. The liver abscesses are to be opened as soon as discovered.

CASE XXIX. *Acute amœbic colitis; death.* Female, 50 years old, Dec. 15, 1894.

One week ago she began to have diarrhœa, about ten movements a day. The stools were large and small, composed principally of serum. She vomited frequently.

Now, emaciated and feeble; temperature 100°; sonorous and subcrepitant rales over both lungs, four watery stools.

Dec. 16. Temperature 99°; nine stools.

Dec. 18. Temperature 102°; died.

Autopsy. The lungs were emphysematous, the bronchi were congested and contained muco-pus.

The left ventricle of the heart was dilated with old thickening of the mitral valve.

The spleen was large.

The kidneys were small and nodular,.

The liver was normal.

The lower part of the small intestine was congested and studded with small ulcers not belonging to the lymphatic glands.

The whole length of the colon was studded with small ulcers with overhanging edges, involving only the glandular coat. In other places large areas of the glandular coat were destroyed. In some places a layer of fibrin coated the bare connective-tissue coat. There was an extensive cell growth in the connective-tissue coat and groups of amœbæ.

CASE XXX. *Acute amœbic colitis; death.* Male, 25 years old, Nov. 21, 1900.

Thirteen days ago, three days after landing from a voyage from Ireland, he was suddenly taken ill with headache, chilliness, vomiting, diarrhœa and prostration. He has continued to have frequent, loose movements, with cramps and tenesmus, and to become sicker.

Now his tongue is red and dry, temperature 102°, pulse 100, no physical signs over the chest or abdomen.

Nov. 23. He occasionally vomited green fluid. In the twenty-four hours he had six stools, composed of mucus, serum and a little fecal matter. He continued to have temperatures from 98° to 100°, five or six passages every day of mucus, serum and a little fecal matter with latterly a little blood. He vomited nearly every day and became steadily thinner and weaker. The treatment with castor oil, opium and whisky had no effect.

Dec. 2. He died.

Autopsy. The autopsy was incomplete.

The small intestine was normal.

The whole length of the colon was studded with large, deep amœbic ulcers, the mucous membrane between was swollen.

CASE XXXI. *Amœbic colitis, abscess of the liver.* Male, 27 years old, Jan. 11, 1895.

Ever since he was a boy he has had attacks of diarrhœa, frequent small stools with burning and tenesmus. Between the attacks the bowels have been constipated.

Two weeks ago he had a chill, felt feverish, with pain and griping in the lower part of the abdomen. Since then he has had diarrhœa, at first three stools a day, then increasing, until on January 10, he had twenty. Three days ago there was blood in the stools. He has vomited every day.

Now, temperature 100°; the stools are composed of yellow, fluid fæces, studded with small lumps of mucus. He is too sick to examine.

Jan. 13. He died.

Autopsy. The bronchi contained muco-pus and the lungs were œdematous.

The heart was normal.

The spleen was large.

The kidneys were large, the cortex pale.

The right lobe of the liver was occupied by an abscess containing a quart of thick pus.

The stomach was distended with food.

The small intestine was normal.

For the entire length of the colon the mucous membrane was blackened and studded with small ulcers. The changes were very extensive. Where there was any glandular coat left there was a growth of fibro-cellular tissue between the tubules and cells in the connective-tissue coat. Necrosis had involved a large part of the glandular coat and the superficial portions of the connective-tissue coat. There were some of the ordinary ulcers with overhanging edges, but for the most part it was a diffuse necrosis.

CASE XXXII. *Acute amœbic colitis, abscesses in the liver and lung.* Male, 51 years old, June 7, 1893.

May 25. He began to have a cough with bloody and foul smelling sputum. He lost flesh and strength rapidly. His bowels were constipated until ten days ago. Then he began to have several loose, watery passages every day without blood or pain.

Now he is feeble and emaciated; three loose movements a day without blood or mucus; cough, with large quantities of brown, foul smelling sputum; temperature 100°; urine contains sugar.

He continued in the same condition, losing flesh and strength; temperature 99° to 101°.

June 18. He died.

Autopsy. The lower surface of the left lung was adherent to the diaphragm. At this point the diaphragm was perforated, there were small abscesses in the lung and below the diaphragm.

The left ventricle of the heart was a little dilated, the valves were normal.

The liver was large, an abscess in the left lobe 2½ inches in diameter, two small abscesses in the right lobe.

The kidneys were large and smooth.

The colon for its entire length was thickly studded with large and small ulcers and sloughs. The most noticeable feature was the necrosis, which extended down to the muscular coat, with but few pus cells, no fibrin and but little cell growth. Amœbæ could be seen in the floors and walls of the ulcers.

3. *The Sub-acute Cases.*

In these cases there is at first simply a diarrhœa, either fecal or watery, with gradual loss of flesh. After a time mucus and sometimes blood are added to the discharges and the stools become larger.

The mild cases may only last a few weeks and then apparently recover, but they are liable to the amœbic abscesses in the liver. In some of the liver cases the history of the colitis is so short and unimportant that it is easily overlooked.

But one such mild attack may be followed by others that are more severe.

In the severe cases the diarrhœa continues, the loss of flesh and strength are progressive and, either with or without abscess of the liver, the patients finally die. It differs from other forms of colitis in the large quantity of semi-fluid fecal matter that is discharged in each twenty-four hours.

CASE XXXIII. *Sub-acute amœbic colitis.* Male, 30 years old, Feb. 17, 1894.

Five days ago he began to have cramps in the right leg, which grew worse, until on February 16 he could hardly walk. The pain involved the whole of the leg and was continuous. There was no swelling, tenderness or redness.

Feb. 17. Since admission there has been no more pain in the leg and there are no symptoms except a fecal diarrhœa and a temperature of 103°. There are no physical signs of the heart, lungs or abdomen.

Feb. 18 to 23. Temperatures 99° to 102°; every day six to twelve loose, fecal passages with a little blood.

Feb. 24 to March 5. Temperatures 99° to 103°; the stools continue frequent, contain less fecal matter and more blood. He is steadily losing flesh and strength.

March 6 to 16. Temperatures 98° to 101°; the diarrhœa continues; the blood now appears in clots of some size. The anterior abdominal wall is flat and rigid, with some tenderness over the colon.

He went on in the same way, and died March 22.

Autopsy. Only the abdomen was examined.

The spleen and kidneys were normal.

The liver was fatty.

The small intestine was normal.

The whole length of the colon was studded with large ulcers, some with overhanging edges, some clean down to the muscular coat, with little islets of mucous membrane between them. There seems to have been a suppurative and necrotic inflammation of the connective-tissue coat undermining the glandular coat.

CASE XXXIV. *Sub-acute amœbic colitis, abscess of the liver.* Male, 46 years old, Nov. 21, 1898.

About twenty months ago he began to have diarrhœa, many large, watery passages daily. This continued for about nine months; he lost much flesh and strength.

Then he seemed to recover entirely. About four months ago the diarrhœa returned, with loss of flesh and strength.

For two weeks he has had occasional vomiting.

Now he is pale, emaciated and feeble; temperature 98°, pulse 104, urine 1.015; trace of albumen.

The liver extends down to the umbilicus, is firm and smooth. The stools are about four a day, composed of blackish, semi-fluid, fecal matter without mucus.

I thought he had an amœbic colitis, an old abscess of the liver with waxy degeneration, and that he was getting worse from the liver, rather than from the colon.

Dec. 2. He died.

Autopsy. There was a thrombus in the left pulmonary artery. There were miliary tubercles in the left pulmonary pleura.

There was a little old thickening of the mitral valve of the heart.

The spleen was waxy.

The kidneys were large, white, waxy.

In the right lobe of the liver there was a large abscess, which had ruptured downward into the abdominal cavity, making a sacculated abscess extending downward around the right kidney. The liver tissue was waxy.

The stomach was dilated; there were many small, superficial ulcers of the mucous membrane.

The pancreas was atrophied.

The mucous membrane of the small intestine was congested.

The whole length of the colon was studded with small ulcers.

The changes in the colon were more marked than one would suppose from its gross appearance.

There was a very considerable diffuse growth of connective tissue between the glandular tubules, and over considerable areas the tubules had disappeared altogether.

There was a rather dense thickening of the connective-tissue coat.

The ulcers were small and not deep, but with overhanging edges.

There were amœbæ in the connective-tissue coat.

VI. IRREGULAR FORMS OF COLITIS.

There are cases of colitis that I am unable to classify. I give the histories of a few such cases.

Case XXXV. *Chronic colitis with very large, white fecal movements.* Male, two and one-half years old, born and living in Brunswick, Georgia.

In July, 1874, he gradually became restless and fretful, with a swollen abdomen. He had one passage a day of undigested food, not large or white. He had a constant but irregular rise of temperature. The movements of the bowels gradually increased in frequency up to six in the tweney-four hours; they became large and white. There was constant thirst and hunger, emaciation and swelling of the abdomen.

November 27. He was brought to New York. I found him very thin and feeble, unable to walk, constantly crying, the abdomen much swollen. There were five movements in the twenty-four hours. Each movement was white and pasty, looking like soft fæces, and so large as to fill a large chamber pot.

He was ordered chops, toast, milk and cod liver oil.

Up to December 8 there was no improvement. Then he was put on a combination of arsenic, hyoscyanus, cubebs and opium. He soon began to improve and continued to do so until January 1, when the opium was stopped.

He continued steadily to improve, and on January 8 the mixture was replaced by dilate nitro-muriatic acid.

Jan. 20, 1875. He is now strong enough to walk. The passages are smaller, less frequent and becoming yellow.

By March 1 he was well.

He continued well for two years, when he was killed in a railway accident.

Case XXXVI. *Purulent inflammation of the colon and stomach.* Male, 43 years old, July 22, 1873.

He was habitually intemperate and had been on a spree for two weeks. This was followed for two weeks by cramps and diarrhœa.

July 20. He went to bed.

The cramps and diarrhœa continued until he died, on July 24.

Autopsy. Ten hours after death.

There was a diffuse congestion of the stomach, and a marked infiltration of pus cells between the gastric tubules.

The small intestine was normal.

The colon for its entire length was congested, roughened and ulcerated. Where the tubules were left the spaces between them were filled with pus cells. The submucous connective tissue is thickened and infiltrated with pus cells.

CASE XXXVII. *Acute entero-colitis, malaria.* Female, 27 years old, October 5.

Two weeks ago she was attacked with vomiting, followed by a severe chill lasting for three hours, then fever and sweating. These paroxysms occurred every day for nine days.

Three days after the first attack she began to have diarrhœa. The diarrhœa, chills, fever and sweating continued up to the time of admission.

She continued in the same condition until she died, on October 9.

Autopsy. The brain, heart and lungs were normal.

The liver and spleen were blackish, with malarial pigment.

The stomach and kidneys were normal.

The colon throughout was congested, its wall was thick, soft and boggy; there were ulcers of the glandular coat; there was fibrin on the peritoneal coat.

In the lower part of the small intestine there were several isolated portions, a few inches long, with lesions of the same character as those in the colon.

No emboli or thrombi could be found.

CASE XXXVIII. *Necrotic colitis, peritonitis without perforation.* Male, 35 years old January 20.

Four weeks ago he began to have diarrhœa, but continued at his work as an engineer until four days ago.

Then he had three large stools of blood, and fainted.

Jan. 22. He had a large stool of blood.

Jan. 23. He died.

Autopsy. There was a little fibrous induration and a small cavity at the apex of the right lung.

The abdomen contained a large quantity of purulent serum.

In the rectum and colon there were many large, irregular ulcers, extending down to the peritoneal coat, without perforation.

In the lower part of the ileum there were a few small ulcers.

The mesenteric glands were swollen.

The kidneys were large; advanced chronic nephritis.

The liver was fatty.

CASE XXXIX. *Necrotic, probably croupous colitis, large bleeding; death.* Female, 35 years old, September 19.

Two weeks ago cramps in the stomach and vomiting. The next day numerous small bloody passages, headache, thirst.

These passages continued and then were succeeded by dark, fluid, partly fecal passages without blood. Pulse 100, temperature 100°.

Sept. 22. a large bleeding from the bowels.

She continued to have small, dark, partly fecal passages until she died on Sept. 28.

Autopsy. The heart was normal. There were old adhesions over the lungs.

The liver, spleen and kidneys were normal. The colon for its entire length shows only patches of mucous membrane left, the glandular coat has nearly all been destroyed.

Case XL. Colitis with abscesses of the liver. Male, 37 years old. Oct. 1.

One week ago he began to have diarrhœa, pain in the abdomen and nausea. Now he is emaciated, pain in the right hypochondrium, face drawn and anxious, skin cold and sweating, tongue dry and brown. He has fulness and tenderness over the right iliac fossa, numerous dark, foul, fluid passages, pulse 100, breathing 46, temperature 100°.

The brown, fluid passages continued, eemperature 98° to 99°, he looked more and more septic and died Oct. 4.

Autopsy. The mitral valve was thickened. The left pleural cavity was one third full of serum, the right pleural cavity was nearly full of serum, the right lung was compressed and coated with fibrin.

The peritoneum was coated with pus and fibrin. The spleen was large and soft. The kidneys were of medium size with adherent capsules.

The liver was large. There were several large abscesses with sloughing walls in the right lobe. The corresponding branches of the portal vein were filled with red and white thrombi.

The caput coli was dilated. Its wall was thick, soft and black, with several large, black sloughs of the entire thickness of the wall. Below this through the whole length of the colon there were numerous small ulcers as if of the solitary glands.

CASE XLI. *Acute purulent colitis.* A Chinaman with imperfect history.

Four weeks ago he was seen by a missionary, who supposed he was well. A friend says that he was sick for about three weeks with dyspnœa, cough and œdema of the feet. Four days before death he came into the hospital, urine 1.019, trace of albumen, granular casts; double aortic murmur, jaundice, profuse diarrhœa, temperature 98° to 100°.

Autopsy. There were three pints of bloody serum in the peritoneal cavity and a few ounces in the pericardial and pleural cavities.

The stomach and small intestines were congested.

The colon was studded with elevated patches about half an inch in diameter. At these points there was a marked purulent infiltration of all the coats of the colon.

CASE XLII. *Purulent colitis.* Female, 26 years old, Nov. 20, 1895.

Two years ago she had a child; she has not been strong since that time.

Sixteen months ago she began to have a diarrhœa which has steadily become worse, five to twelve stools daily, large and watery with some mucus but no blood.

When she eats she has griping pains in the abdomen, no nausea or vomiting, but not much appetite. She has lost flesh and strength, but was up and about until a month ago. Now, the tongue is red, glazed and clean, temperature 100°, pulse 108, she is feeble and emaciated. She has 4 to 12 stools daily, large watery, containing but little fecal matter, some small lumps of mucus, no blood. The stools continued the same, she became weaker and died Nov. 27.

Autopsy. Only the colon was examined. It contained no fecal matter. For its entire length it looked as if it was coated with a thin layer of fibrin, no ulcers could be seen. Minute examination shows that the mucous membrane is coated with pus cells, not with fibrin.

The glandular coat is thickly infiltrated with pus cells, in some places extending into the connective-tissue coat.

There are many small erosions of the glandular coat where the tubules are broken down and replaced by pus cells.

There is also a growth of connective-tissue cells between the glandular tubules.

CASE XLIII. *Necrotic Colitis of unknown character.* Female, 48 years old, Dec. 19, 1894.

For several months she has been losing flesh.

Three days ago she began to vomit coffee-ground matter.

Now she is very feeble, temperature 96°, pulse 160, vomits coffee-ground matter.

Dec. 20, she died.

Autopsy. The bronchi were congested and coated with mucous.

The mitral valve was thick and the left ventricle of the heart hypertrophied. The spleen was normal.

The kidneys were large, congested and studded with small abscesses.

The stomach was small, contained brownish fluid, but looked normal. The liver showed the lesions of cirrhosis, the gall bladder was distended with bile.

The pancreas and small intestines were normal.

The urinary bladder was normal. The upper part of the colon was intensely congested. It was studded with areas where the mucous membrane was black and swollen. In these areas the blood vessels were distended with blood, the glandular coat was necrotic, the connective-tissue coat was infiltrated with pus cells.

CASE XLIV. *Chronic Colitis.* Female, 27 years old, May 31,

Two months ago her ankles were swollen and there was occasional vomiting. She began at that time to have a number of passages of mucus, sometimes mixed with blood, every day.

These passages gradually increased in number, she lost flesh and strength, and without any other symptoms died on June 10.

Autopsy. The heart, lungs and liver were normal.

The kidneys showed the lesions of chronic nephritis.

The spleen was covered with old adhesions.

The stomach was pale and coated with mucus.

The small intestine was normal. In the colon for its entire length there were large superficial ulcers. The connective tissue and muscular coats were thickened. In the lower part of the colon there were polypoid thickenings of the mucous membrane.

CASE XLV. *Old productive, recent purulent colitis.* Female, 30 years old.

She was admitted to the hospital as a case of abortion with a temperature of 101°. She had a foul discharge from the vagina and when the temperature reached 103° she was curetted.

She stood the operation badly, but after 12 hours was in pretty good condition.

For 24 hours after this the temperature was subnormal, then it got up to the normal but did not go above 99°.

Before the operation the bowels moved once a day, the stools were not abnormal. An enema was given before the operation and came away blood stained.

For four days before death she had from ten to fifteen stools a day. The stools were small and composed of bloody mucus.

Autopsy. The cortex of the brain was intensely congested.

The spleen, liver and kidneys were large and soft.

The uterus with its appendages and the peritoneum were normal.

The lungs were congested and œdematous. The lower half of the colon on its inner surface was thrown into irregular elevations of considerable thickness and of grayish color.

Vertical sections show that the glandular coat has everywhere disappeared.

The inner edge of the bare connective coat is in some places infiltrated with pus cells and coated with fibrin.

The elevations are formed of a new formation of rather dense connective tissue.

CASE XLVI. *Old colitis, perforation of the colon.* Female, 43 years old, May 16.

The patient says she has always had some weakness of the bowels, having slight diarrhœa after exposure to cold, but that she has never been sick.

On May 14 she ate some food that disagreed with her and that night she had abdominal pain and several loose passages.

On the night of May 15 she had 10 loose passages.

On the night of May 16 and morning of the 17 she had three loose, foul, fecal stools without pain or tenesmus.

After May 19 the diarrhœa stopped. May 23, temperature 100° to 102°. May 24, temperature 102°, tongue dry, delirious, rapid pulse, increasing distension of the abdomen.

In the evening she died.

Autopsy. The peritoneal cavity was empty and the peritoneum did not look imflamed.

In the small intestine the solitary and agminated glands were swollen. The descending colon was the seat of old and extensive ulceration involving the glandular and connective-tissue coats. At the junction of the middle and lower thirds there were two perforations of the peritoneal coat, each about one quarter of an inch in diameter. The edges were adherent to the anterior abdominal wall with a little recent fibrin.

The other viscera were normal.

CARCINOMA OF THE COLON.

The new growth usually follows the type of colloid cancer, or of glandular cancer with cylindrical cells. The favorite situations are the rectum and the caput coli. The new growth surrounds the gut and extends longitudinally for one or two inches; less frequently it involves a considerable part of the length of the colon. The growth may cause a stricture of the colon, or may project inward in soft masses, or may ulcerate. It often infiltrates the surrounding tissues, it may cause a local peritonitis, or the ulceration may go on to perforate the wall of the colon.

Causes.—The disease occurs regularly in persons over thirty years of age.

Symptoms.—If the growth is situated in the rectum the first symptoms are constipation, flatulence, pain, and constricted stools; or simply a number of small fecal passages every day. The patients complain of the annoyance of having to defecate so frequently instead of having a single large movement. In other patients there is at first a diarrhœa, the passages are fluid, and more or less blood and mucus are also passed. At first, and even for a number of months, the general health suffers very little.

As the disease continues the local symptoms become more marked, and the patients lose flesh and strength and become cachectic. The tumor can be felt in the rectum as a hard ring constricting the gut, or as a hard diffuse mass about the gut, or as a soft tumor projecting inward. As the disease goes on the stricture becomes tighter and tighter; or there is paralysis of the sphincter with a constant flow of brown, sanious fluid.

The patients die worn out with the disease, or with retention of fæces, or from perforation of the intestine, or from peritonitis.

If the growth is situated in the caput coli the early diagnosis is often difficult. Until a tumor can be felt it is easy to be mistaken.

The first symptom may be only attacks of abdominal pain first at long intervals then more and more frequent.

Or there may be flatulence, alternating diarrhœa and constipation, nausea and vomiting.

Or there may be simple loss of flesh and strength without any local symptoms.

Or the symptoms of obstruction are prominent, a gradual distension of the small intestine with vomiting.

The patients in whom there are no marked symptoms until there is a sudden, complete stoppage of the bowels are very misleading.

The only treatment is surgical.

CASE XLVII. *Colloid cancer of the rectum, rupture of the colon, in a young man.* Male, 19 years old, Feb. 19.

Seven months ago he was attacked with diarrhoea and vomiting, about ten stools a day.

After two weeks he went to work again, but the diarrhœa continued and after two months he was obliged to stop work. The passages were composed of mucus and blood and were painful. For the past two months the passages have been involuntary.

For the past two weeks there have been from ten to twenty passages a day with more blood.

There is now uniform distension and tenderness of the whole abdomen.

March 6 he died.

Autopsy. There were no changes in the brain, heart, lungs, liver, spleen or kidneys.

The peritoneal cavity contained a large quantity of fluid fæces, but there was no peritonitis.

The entire colon was enormously distended with fluid fæces. At the splenic flexure there was a large rupture.

In the rectum two inches above the anus there was a mass of new growth as large as a man's fist. It completely surrounded the rectum. It involved all the coats of the gut, but grew outward; there were no projections into the cavity of the rectum. The caliber of the rectum was narrowed, but not occluded. The posterior wall of the bladder was involved in the new growth. The growth has the structure of colloid cancer.

CASE XLVIII. *Carcinoma of the descending portion of the colon.* Male, 37 years old, July 6.

Nine months ago he began to have pain in the left side with diarrhœa and was confined to bed.

Five months ago he had a similar attack and then vomited coffee-ground matter.

For the past two months he has lost flesh and strength with vomiting, flatulence and severe pain in the left side.

Now, he is feeble, emaciated and anæmic. There is profuse vomiting. The abdomen is distended with visible peristalsis of the intestines, no tumor can be felt.

July 28, he died.

Autopsy. The body was much emaciated. In the heart there was a little old thickening of the mitral valve. There were old pleuritic adhesions, emphysema and chronic bronchitis. The stomach showed the lesions of chronic gastritis. The liver and spleen were normal.

The kidneys were atrophied, chronic nephritis. The small intestine was dilated, its glandular coat thickened and ulcerated in irregular patches.

The ascending, transverse and first half of the descending colon were dilated. In the lower portion of the descending colon there was a large ulcer surrounding the gut, three inches long with thickened edges. The thickened edges had the ordinary structure of cylindrical celled carcinoma.

CASE XLIX. *Carcinoma of the caput coli with general anasarca.* Female, 40 years old, Aug. 28.

For a year, she says, she has had cough, expectoration and night sweats. Seven weeks ago the legs became œdematous and then general anasarca supervened.

For five weeks she has had diarrhœa.

Now, she is emaciated, with general anasarca and loose bowls.

Sept. 3 she died.

Autopsy. There were old adhesions over both lungs. The heart was small and flabby.

The liver was fatty.

The kidneys were of medium size, chronic nephritis, not far advanced.

The stomach and small intestine showed thickening of all their coats. In the caput coli there was a large, fungous mass growing inwards, but not constricting the gut. It was composed of cylindrical cells in tubules.

CASE L. *Colloid cancer of the caput coli.* Male, 54 years, March 18.

Two months ago he gradually began to lose flesh and strength and had to give up work. He had nausea, vomiting, pain after eating and flatulence.

Now, he is feeble and emaciated, a small tumor can be felt in the right iliac region.

April 21 he died.

Autopsy. There was recent fibrin over the lower lobe of the left lung, general bronchitis and broncho-pneumonia of both lower lobes.

The heart was small.

The liver was small and fatty.

The spleen and kidneys were normal. The wall of the caput coli was infiltrated with a soft, colloid growth forming a tumor the size of a turkey's egg.

The small intestine above this was dilated, congested and ulcerated.

CASE LI. *Lymphoma of the caput coli and mesentery.* Male, 70 years old, Dec. 16.

Eight years ago he noticed a swelling in the right groin.

One year ago he found a tumor in the right side of the abdomen in the situation of the caput coli. The bowels have been constipated, he has lost flesh and strength.

Now, he is thin and anæmic. There is a tumor in the abdomen about the caput coli and extending to the left. Diagnoses were made of lymphoma of the mesentery, retro-peritoneal sarcoma and carcicoma of the caput coli.

January 7. *Autopsy.*

There is a large mass of enlarged glands in the mesentery and a smaller mass of retro-peritoneal lumbar glands.

The wall of the caput coli is thickened in places as much as one and a half inches and infiltrated with new growth. The cavity of the caput coli is enlarged, patches of new growth lift up the glandular coat. The growth has the anatomical character of lymphatic glandular tissue.

CASE LII. *Lymphoma of the jejunum simulating a tumor of the caput coli.* Male, 19 years old.

In May he had an attack of fever lasting four weeks, that was called malaria. After this he had persistent diarrhœa.

October 8 he was admitted to the hospital with a tumor in the right iliac fossa.

October 12 the tumor was aspirated and two drachms of pus withdrawn.

October 13 the abscess was opened and some fecal matter escaped.

October 29 he developed symptoms of peritonitis and died after a week.

Autopsy. There was general peritonitis.

The intestines were matted together.

The incision of the operation was opposite the cicatrix of an opening in a distended portion of the jejunum. The distension was produced by a new growth.

The condition of the intestine was peculiar. About one foot of the jejunum was dilated and was fastened by adhesions in the position of the caput coli, so that at first it was thought to be the latter. The wall of the dilated portion of intestine is very soft and there are many deep ulcers nearly through the peritoneal coat. There are also flattened tumors in the wall and on the attached side. These tumors are large and continuous with a mass of enlarged mesenteric glands so as to form altogether a mass of some size. All the tumors have the structure of lymphatic glandular tissue.

Case I.—Chronic constipation.

Case II.—Insufficient production of fæces, long intervals without a movement from the bowels.

Case III.—Retention of fæces simulating peritonitis.

Case IV.—Retention of fæces simulating peritonitis.

Case V.—Retention of fæces simulating diarrhœa, death.

Case VI.—Retention of fæces simulating diarrhœa, death.

Case VII.—Retention of fæces simulating typhoid fever.

Case VIII.—Acute catarrhal colitis.

Case IX.—Acute catarrhal colitis.

Case X.—Acute catarrhal colitis with serum.

Case XI.—Acute catarrhal colitis with serum.

Case XII.—Chronic colitis with mucus.

Case XIII.—Alternating constipation and diarrhœa with large discharge of mucus.

Case XIV.—Chronic colitis with mucus.

Case XV.—Chronic catarrhal colitis, death.

Case XVI.—Chronic catarrhal colitis, death.

Case XVII.—Productive and necrotic colitis, death.

Case XVIII.—Productive and necrotic colitis, death.

Case XIX.—Productive and necrotic colitis, death.

Case XX.—Chart 1. Productive and necrotic colitis, death.

Case XXI.—Chart 2. Productive and necrotic colitis, recovery.

Case XXII.—Productive and necrotic colitis, death.

Case XXIII.—Croupous colitis, death.

Case XXIV.—Croupous colitis, death.

Case XXV (a).—Croupous colitis, recovery.

Case XXV (b).—Croupous colitis, recovery.

Case XXVI.—Croupous colitis, death.

Case XXVII.—Ulcers after croupous colitis.

Case XXVIII.—Acute amœbic colitis, recovery.

Case XXIX.—Acute amœbic colitis, death.

Case XXX.—Acute amœbic colitis, death.

Case XXXI.—Amœbic colitis, abscess of the liver.

Case XXXII.—Amœbic colitis, abscesses in the liver and lung.

Case XXXIII.—Subacute amœbic colitis, death.

Case XXXIV.—Subacute amœbic colitis, abscess of the liver.

Case XXXV.—Chronic colitis with very large, white fecal movements.

Case XXXVI.—Purulent inflammation of the stomach and colon.

Case XXXVII.—Acute entero-colitis, malaria.

Case XXXVIII.—Necrotic colitis, peritonitis without perforation.

Case XXXIX.—Colitis—probably croupous—large bleeding, death.

Case XL.—Colitis with abscesses of the liver.

Case XLI.—Acute purulent colitis.

Case XLII.—Purulent colitis.

Case XLIII.—Necrotic colitis.

Case XLIV.—Chronic colitis.

Case XLV.—Old productive, recent purulent colitis.

Case XLVI.—Old colitis, perforation of the colon.

Case XLVII.—Colloid cancer of the rectum, rupture of the colon.

Case XLVIII.—Carcinoma of the descending colon.

Case XLIX.—Carcinoma of the caput coli.

Case L.—Colloid cancer of the caput coli.

Case LI.—Lymphoma of the caput coli.

Case LII.—Lymphoma of the jejunum.

THE STOMACH.

Disturbances of the Functions of the Stomach without Organic Disease. Gastric Dyspepsia.

Disturbances of the functions of the stomach without structural disease of its walls are produced in a variety of ways.

1. The habitual ingestion of food in excessive quantities, or of improper quality, may after a time cause gastric symptoms. It must, however, be remembered that gastric symptoms are often due to entirely different causes, and that too little food is as bad as too much.

One of the simplest disturbances due to an excessive ingestion of food is seen in children. A child at some particular meal overfills its stomach. The pylorus remains closed and no gastric digestion takes place. After some hours the child has abdominal pain, rapid heart action, a cold skin, and sometimes marked prostration. Finally it vomits the food in nearly the same condition as when it was taken into the stomach, and is at once relieved of all symptoms.

The same thing can be seen in adults. A large meal is eaten, little or no gastric digestion takes place, the pylorus remains closed, the stomach is distended with undigested food. After a number of hours the patient has the feeling of vertigo or of faintness, or he may fall to the ground unconscious. Then there is a profuse vomiting of the contents of the stomach.

In adults habitual over-eating may cause chronic gastritis and dilatation of the stomach, but it is much more apt to cause symptoms referable to disturbances of the functions of the liver —feelings of lassitude and depression, headache, constipation, diarrhœa, flatulence, an excess of urates in the urine.

The habitual eating of improper food results in an impairment of nutrition, for not only is the improper food bad in itself, but it takes the place of wholesome food.

The treatment of these cases consists in a regulation of the diet. In the bad cases the patients must be put for a time on the exclusive use of milk, or of scraped beef, but neither of these exclusive diets can be continued with advantage for any great length of time. The use of all sorts of health-foods, peptonized foods, pepsin and pancreatine, is to be avoided. The patients must be educated to eat meat, starches, fats, vegetables, and fruits in proper quantities. In doing this the use of the stomach-tube, about four hours after one of the meals, is of much service as a guide to the particular articles of food which are best digested and remain the shortest time in the stomach.

2. Changes in the gastric juice. The two most marked changes in the gastric juices are: such a change in its composition as renders it unable to perform stomach digestion, or increased quantity and hyperacidity.

(a) Changes in the composition of the gastric juice of such a character that stomach digestion is imperfectly performed.

This condition is seen in persons who have had chronic gastritis for a long time, with changes in the glandular coat of the stomach. It is also seen in young persons in whom there is no evidence of disease of the glandular coat.

The patients have no appetite, or even a distaste for food. In some there is occasional nausea, vomiting, and pain after eating. The bowels are usually constipated. The nutrition suffers noticeably, the patients lose flesh and strength. Besides the gastric symptoms there are often a variety of accessory symptoms of nervous and hysterical character. It is not uncommon to find persons belonging to this class who do not complain of any gastric symptoms, but only of the loss of nutrition and some disturbance of sensation.

On the other hand, we must not confound with this class the persons who complain of gastric pain and oppression, loss of appetite, and nausea, but in whom gastric digestion is well performed.

The diagnosis is made with the help of the stomach-tube.

There will be found a considerable number of persons in whom more or less partly digested food is always present in the stomach. The problem in these persons is to improve gastric digestion. It would seem as if this could be done by the use of pepsin, or hydrochloric acid. or partly digested foods, but it will be found that no permanent improvement can be effected in this way. There is no one rule for the management of all these patients, but the general plan is to improve the general health by massage, baths, exercise, and travel, and to feed the patients as well as can be done. The method of feeding has to be found out for each person with the stomach-tube. We try the simple articles of food—milk, eggs, koumyss, meat, beef-juice, gruels, etc,—and see which ones leave the cleanest stomachs in the shortest time. In this way we get a guide as to the best kind of food and the proper intervals between taking food. As the patients improve we gradually add other kinds of food. It requires a number of months to get these persons back to a natural condition of health.

(b) The gastric juice may contain an excessive quantity of hydrochloric acid, and be itself produced in too large quantities. The most marked symptoms of this condition are epigastric pains and vomiting. To determine this condition accurately the stomach should be washed out in the evening and its contents aspired the next morning before the ingestion of food or fluid.

The milder cases are benefited by the use of alkalies, but the only satisfactory treatment is the systematic washing out of the stomach.

Temporary insufficiency of the gastric juice may be caused by a variety of nervous and mental conditions.

3. The muscular coat of the stomach may cease to perform properly its function of moving and expelling the food. This condition is apt to be slowly developed, so that it is not until after several years that it becomes a serious evil. The insuffi-

cient action of the muscular coat of the stomach allows food to remain in the stomach from one meal to another, so that the organ is never empty. This constant presence of food may eventually cause chronic gastritis or dilatation of the stomach. The symptoms at first come on in attacks which last for several days; between the attacks the patient is well. As time goes on the attacks become more frequent and of longer duration, and finally the symptoms are continuous. The principal symptom is pain of greater or less severity referred to the region of the stomach. To the pain are regularly added after a time constipation, nausea, vomiting, and finally loss of flesh and strength.

During the earlier periods of their trouble the patients improve under an absolute diet of milk or of meat, but the only really satisfactory treatment is the washing out of the stomach, with a regulated diet.

4. Disorders of the colon—constipation, chronic inflammation, or carcinoma—may be accompanied by gastric symptoms—pain, nausea, and vomiting. In elderly persons, whenever nausea and vomiting are present, it is always necessary to think of an accumulation of fæces in the colon as the probable cause.

5. Diseases of the uterus, ovaries, and tubes are rather frequently accompanied by loss of appetite, nausea, and vomiting. These symptoms are continuous, or come on in attacks. There may really be chronic gastritis, or inefficient gastric juice, or retention of food in the stomach in addition. But it will be found that the treatment of the stomach is unsatisfactory unless the disease of the uterus, ovaries, or tubes, can first be cured.

6. In the simple anæmia of young women gastric symptoms are usually present, and in some of the patients are especially severe. Loss of appetite, nausea, and vomiting are often present, hæmatemesis is not rare. The gastric symptoms may be so marked as to make it easy to overlook the anæmia, but this error is readily avoided by the examination of the blood.

In these anæmic patients the gastric symptoms promptly disappear with the cure of the anæmia.

7. Hysterical patients are capable of developing a great variety of gastric symptoms—loss of appetite, perverted appetite, distaste for food, nausea, vomiting, pain, and flatulence.

As a rule these patients are best treated if we disregard the gastric symptoms and employ the regular management for hysterical cases.

8. The habitual use of tobacco and of alcohol will, in some persons, even before chronic gastritis is produced, cause loss of appetite, nausea, and vomiting.

9. Neurasthenia. Of the patients who suffer from the condition of neurasthenia a considerable number have decided gastric symptoms. Generally speaking, it is better not to pay too much attention to the stomach, but to rely on baths, massage, exercise, climate, etc. I see neurasthenics, however, who are entirely relieved by lavage of the stomach. This seems to be a simple faith cure.

10. In some persons gastric symptoms are produced by a movable kidney.

This is often associated with displacement of the stomach downward. It is a curious fact that in many of these patients the gastric symptoms may entirely disappear although the displacement of the kidney and stomach persist.

CASE I. *Distress after eating and vomiting without disease of the stomach, recovery.* Female, 50 years old, twice a widow, no children, no occupation, with a comfortable income. January, 1903.

Several months ago she began to pass small quantities of mucus with the fæces, but had no other symptoms.

For the past three months very soon after eating, without nausea, she vomits part of the food and a considerable quantity of mucus. When she does not vomit she has distress for several hours after eating. Her general health and weight have remained unchanged and she now looks fat and well.

Physical examination shows no changes in any of the viscera. The blood is normal.

Lavage, three hours after a meal of milk, bread and eggs, shows some undigested food and mucus.

The stomach was washed out for two or three days, after that she took an alkali and a bitter for a couple of weeks. She was allowed to eat meat, vegetables and fruits. A certain degree of moral effect was produced. After a month there were no more stomach symptoms. She has continued to have good health for three years.

CASE II. *Stomach symptoms without real disease.* Male, 30 years old, Jan. 27, 1903.

His own statement of his case was as follows:

"Food sometimes makes me feel sluggish and heavy. I have no relish for my meals and eat very lightly. I have a sensation most of the time of having undigested food in my stomach and intestines. There is usually a feeling of depression at meals, so much so that I hesitate to dine out. Quite often there is a full feeling of the head at meals with flushing of the face. Sometimes there is no discomfort at meal time but a feeling of nervous excitability two hours afterwards."

He went through a variety of cures and finally took the hunger cure, that is, he was only to eat when he was hungry. On this cure he went for six days at a time without food.

When I saw him he was feeble and emaciated, the stomach and intestines were distended with gas, after a meal of milk, eggs and bread the stomach washed clean.

He was simply put on two full meals a day, and after that had no more trouble.

CASE III. *Symptoms resembling those of cancer of the stomach, complete recovery.* Female, 45 years old, July 19, 1901.

She has lived for years in a malarial district in the South.

One year ago she began to have attacks of abdominal pain on the left side, with occasional nausea and feelings of fever. The pain was made worse by eating, she had about two attacks a month. The attacks of pain increased in frequency and severity until for two months past the pain has been nearly constant. On account of the pain she has left off one article of food after another until for the past three weeks she has taken only peptonoids. She has only vomited once, she has disgust for food, the bowels have moved pretty well. She has steadily lost flesh and strength.

When I first saw her she was very thin and cachectic looking as if she had malignant disease. The spleen was somewhat enlarged, but otherwise I could feel nothing in the abdominal cavity. The stomach washed clean.

I began by feeding her with small quantities of milk containing oxalate of cerium and bicarbonate of soda. After a week she ate meat once a day.

July 29 she was feeling much better and had no gastric pain.

After this she improved steadily and her food was gradually increased. She had no more gastric symptoms. In January 1902 I saw her and she was very well.

CASE IV. *Gastric symptoms without disease of the stomach.*

The patient's own account of himself was as follows;

"I am forty-two years of age, my earliest recollections are of dyspepsia. At sixteen I entered business life, since then dyspepsia has been constant, with acute attacks and constipation.

"At first there was difficulty in swallowing food, flatulence and headache, this condition continued for many years and for a few years past I have had distress in the back between and below the shoulders, which has been of the most distress and the greatest burden. As a rule the trouble in the back asserts itself most strongly about three hours after eating, when it causes almost complete exhaustion.

"Food is often raised, more frequently two or three hours after midday and evening meals.

"During all these years convulsions have at times overpowered me, generally immediately after lying down at night. For the past six years these have been infrequent, they are now recurring oftener. Numerous remedies have been taken, also skim milk diet for three months, electrical treatment, inhalations of oxygen, constant use of cathartics, flushing the bowels with three quarts of water, drinking hot water before meals. Relief has been only temporary, a constant change being necessary to obtain any relief."

April 3 I first saw him, three and a half hours after a breakfast of coffee, egg and bread; the washing of the stomach gave a little egg and a considerable quantity of mucus.

April 4, after the same breakfast, the stomach washed clean.

April 13, he has been washing the stomach once a day and eating two good meals. The bowels move every day without medicine and he feels well.

He continued to be well for a month and passed out of my observation.

CASE V. *Persistent vomiting without disease of the stomach; recovery.* Female, 43 years old, Jan. 1, 1894.

April 16, 1880, she was said to have had peritonitis.

Sept. 18, 1880, she was said to have had an abscess on the left side. She was unable to retain ordinary food and lived on stimulants and milk.

Feb. 17, 1881, the abscess broke. Since that time she has never gone a week without vomiting some of her food.

July, 1888, severe pains in the stomach for three weeks, constant vomiting, confined to bed.

June, 1891, another similar attack, since that time she has never retained food for two successive days.

Since May 15, 1892, she has lived on bread and a few vegetables and has had pain and vomiting every day. The bowels have continued to move and menstruation has been regular.

Jan. 1, 1894, I saw her for the first time, she was much emaciated, weighing 103 pounds. I could find no evidence of peritoneal adhesions, no tumor, no dilatation of the stomach, the stomach washed clean.

I thought that the vomiting had begun with the abscess in the abdominal cavity, but had continued as a habit.

The patient lived about forty miles away from New York. From January 1 to June 23 I made her come to the city every day, washed out her stomach and then gave her a solid meal. At first her progress was slow and she continued to have pain and vomiting, but after a time there were more and more days without vomiting, and by June she was well, eating three meals a day.

I saw her again in February, 1895. She has had no stomach symptoms since last August, feels perfectly well and weighs 145 pounds.

CASE VI. *Marked gastric symptoms without disease of the stomach; recovery.* Female, 17 years old, Feb. 17, 1900.

She has had dysmenorrhœa for 7 years. She has had every year from one to three attacks of colic, vomiting, headache and sometimes diarrhœa. Each attack lasts 24 hours. Otherwise her health has been good.

Ten months ago she had one of her attacks rather more severe than usual. Following this there was backache, dizziness and insomnia and she did not feel well.

Seven months ago she began to have severe pain after eating with frequent eructation of gas. These symptoms continued until five months ago she took to her bed and soon after suffered with another acute attack of colic.

She was then put on rectal feeding for two weeks, with this the nausea and vomiting continued, but the pain stopped.

Solid food was then resumed and after a week the pain and eructation of gas returned, pepsin and bismuth had no effect. For two months the stomach tube has been used irregularly, chiefly to empty the stomach and relieve pain.

Three weeks ago, as the symptoms were growing worse, rectal feeding was resumed and she was no better. She has also taken iron without benefit. The bowels have been constipated throughout, the appetite pretty good. For four months she has suffered from constant dull pain low down in the left side of the abdomen. She has lost weight to a moderate extent. One week ago the abdomen was examined under ether and nothing abnormal was found.

Feb. 17. She is somewhat emaciated, there are no physical signs over the chest and abdomen, hæmoglobin 77 per cent., red cells 4,792,000, white cells 7,400. She was put on the following diet :

7.30 A. M. Koumyss, 6 ounces.
10 A. M. Scraped beef, 1 ounce.
12 M. Cream, 1 ounce.
2 P. M. Granum, 4 ounces.
4 P. M. Beef juice, 2 ounces.
6 P. M. Koumyss, 6 ounces.
9 P. M. Koumyss, 6 ounces.

The rectum was to be irrigated with two quarts of hot salt solution once a day.

Feb. 19.
7.30 A. M. Tea and toast.
10 A. M. Scraped beef.
12 M. Cream.
2 P. M. Granum.
4 P. M. Beef juice.
7 P. M. Bread and butter and baked apple.

Feb. 24. She began to take three ordinary meals.

Her improvement was uninterrupted and by the end of March she was well.

ACUTE CATARRHAL GASTRITIS.

Lesions.—The changes are the same as in all acute catarrhal inflammations—congestion, swelling, at first dryness, later an increased production of mucus, exudation of serum, emigration of white blood-cells. The inflammation may involve the whole of the glandular coat of the stomach, or only its pyloric portion; it may extend to the duodenum.

Causes.—The disease occurs at all ages and in both sexes. It may occur at any time of the year, but is specially common in summer. It is apparently sometimes caused by atmospheric conditions. It frequently complicates the exanthemata, typhoid fever, and epidemic influenza. It may be produced by unwholesome and irritating food. It is of frequent occurrence in persons who have fatty degeneration of the liver. It occurs very often as a primary inflammation. Some persons show a marked predisposition to the disease, and suffer from repeated attacks. This is especially the case in some young children. They will have one or more attacks of acute gastritis every year for several years. As they grow older the attacks are less frequent and less severe.

Symptoms.—The symptoms of an acute gastritis may come on suddenly or they may be preceded by symptoms of gastric indigestion, of subacute gastritis, or by a diminution of the quantity of bile in the stools.

Pain is often complained of. It may be a severe pain, or only a feeling of soreness or of discomfort.

Vomiting is regularly present. It is in proportion to the severity of the gastritis, and is most troublesome during the first days of the inflammation. The patients vomit whatever they take into the stomach, and more or less mucus and serum. In severe cases large numbers of pus-cells and some red blood-cells are found in the vomited mucus and serum. The irritability of the stomach may be so great that neither food nor medicine can be retained.

A rise of temperature accompanies the first days of the inflammation in some patients, but in many there is not at any time a febrile movement. As a rule, the bowels are constipated. But in some patients an acute colitis is developed at the same time as the gastritis, and then there is diarrhœa. There is a good deal of variety as to the length and severity of the attacks, the frequency of the vomiting, the height of the temperature, and the degree of the prostration. Attacks of acute gastritis are not infrequently followed by subacute and chronic gastritis.

Course of the Disease.—(1) In infants and young children the vomiting is incessant, and excited by everything which is taken into the stomach; the rise of temperature is often well marked, and the prostration is considerable. The gastritis may be accompanied with or followed by diarrhœa. The symptoms may last only for a few days, or for several weeks. Such a gastritis may not at any time be serious, or it may produce a prostration which lasts for weeks, or it may be fatal.

(2) Mild cases in adults. The patients are not at any time very sick, nor is there a rise of temperature. There are loss of appetite, nausea and vomiting, with a feeling of discomfort or of actual pain referred to the stomach. The tongue is coated, the bowels may be constipated. The attack usually runs its course within a week and terminates in recovery, but the inflammation may pass into the subacute or chronic condition.

(3) Severe gastritis in adults is attended with the same symptoms, but they are more marked.

The patients are sick enough to stay in bed, in some the prostration and feeble heart-action are alarming, in some the loss of flesh and strength are extreme. A febrile movement may be present or absent. The feeling of nausea is constant, the vomiting frequent, the pain and discomfort most distressing. Even a severe attack may run its course within a week, but the attacks often last much longer, and may be succeeded by subacute or chronic gastritis.

4. The inflammation involves either the whole stomach, or the pyloric end of the stomach, the duodenum, and the common

bile-duct. The patients then have the symptoms described under the name of simple jaundice.

5. The inflammation is confined to the pyloric end of the stomach and the duodenum. The symptoms are the same as those of an attack of simple jaundice, but without the jaundice. The patients suffer from general malaise, headache, dulness, drowsiness, vertigo. The tongue is coated, loss of appetite, nausea, occasional vomiting, pain in the epigastric region and sometimes fever are present. The bowels are constipated and the fæces light colored.

These cases of pyloric gastritis are sometimes puzzling. The attacks may be repeated during months and years, the patient well between the attacks. There may be no vomiting at all. The rise of temperature may be the principal symptom—up to 103° or 104° F. It may very well happen that these patients are brought to you as examples of an obscure periodic fever without the suspicion that anything is the matter with the stomach.

Treatment.—There can be no doubt that in some persons acute gastritis is caused by hot weather. It is important for such persons to pass their summers in a cool climate.

In infants gastritis is often caused by the food. Not that the food is irritating, but that it contains pathogenic bacteria and the products of their growth. This is an additional reason for the most scrupulous cleanliness in preparing and sterilizing the food of children.

In any case of acute gastritis it is well to keep the patient in bed. For the first twenty-four or forty-eight hours the irritability of the stomach may be so great that it will retain nothing. When this is the case it is wise to desist altogether from giving food or medicine by the mouth. After this the food is given at first by the teaspoonful and the quantity gradually increased. The best foods are usually cream and water, koumyss, milk, beef-tea, beef-juice, and scraped beef. Rectal enemata help but very little in nourishing these patients.

The external application of heat over the upper portion of the abdomen by fomentations, hot-water bags, or poultices is grateful to the patient.

Of drugs the sulphate of morphia and codeia are the most valuable. They are to be given in small doses, one-fiftieth to one-eighth grain of morphia, one-twelfth to one grain of codeia, at intervals of from one to three hours. They are to be used in the form of tablets, which dissolve in the mouth, or given hypodermically.

The hydrochlorate of cocaine is sometimes of use for adults, one-tenth grain in tablet form at intervals of from one to four hours.

Ipecac in tablets of one-tenth grain given every hour may exert a specific effect on the gastritis. The oxalate of cerium and bicarbonate of soda are useful drugs; they are best given in milk or in cream and water. A good formula is to mix together in a half-pint measure equal parts of cream, milk, and water, add to this ten grains of oxalate of cerium and twenty grains of bicarbonate of soda; give a teaspoonful every half hour.

When the gastritis involves the pyloric end of the stomach, rectal enemata of very hot or very cold water, and purgation by calomel, may be of much service.

Case VII. *Acute, followed by subacute gastritis.* Female, 17 years old, living in Chicago.

In October, 1893, she began to have pain and frequent vomiting.

After a month she went to bed with so called "gastric fever." She could retain no food, had constant burning in the stomach and was said to have high temperature. She was in bed four weeks.

Then she improved so that she was up and about, but continued to vomit and has done so nearly every day up to the present time. She has taken liquid peptonoids, Valentine's beef, and many medicines without any improvement.

Sept. 26, 1894, she came to me.

She was fed with milk and soda for a few days, then one solid meal a day, then two meals, then three meals. The stomach was washed out every day.

The vomiting stopped at once. By Oct. 15 she had no gastric symptoms and was eating three meals a day.

CASE VIII. *Acute gastritis, severe form.* Male, 35 years old, school teacher, February, 1902.

One month ago he began to vomit every day—food and green fluid. He also had two or three watery passages from the bowels, with abdominal pain. There was also a rise of temperature and prostration, but he did not go to bed.

The man now looks badly. The stomach washing gives undigested food, yellow fluid and mucus.

The stomach was washed out every day, he was given one meal of meat daily and milk the rest of the time.

This treatment was continued steadily for a month. The fever and vomiting stopped, the bowels were occasionally loose, the stomach washings still contained mucus.

He then went abroad, continuing to wash the stomach.

In June I saw him again, he was well without treatment.

In September he again reported himself as continuing to feel well.

CASE IX. *Pyloric gastritis, repeated attacks.* Male, 45 years old, May 6, 1901.

For a number of years he has been actively engaged in business with too much excitement and not enough sleep. He has had two attacks of nervous prostration, each lasting for two months. Six years ago he gradually became jaundiced and remained so for six weeks, no pain, some nausea, occasional vomiting, marked lassitude.

For the past five years he has had severe attacks of diffuse abdominal pain, lasting from half an hour to several hours and occurring about once a month, sometimes the attacks were repeated on several successive days. Large doses of olive oil relieved the attacks for some time. He now has about four attacks each month.

I thought that the diagnosis was not certain but that it would be wise to treat it as a pyloric gastritis.

From May 6 to June 1 the stomach was washed every day, at first there was a good deal of mucus and undigested food. The pain stopped, the lavage was discontinued.

In October he reported that he had had no more pain and was very well.

March, 1902, after overwork the attacks of abdominal pain recurred.

The stomach was again washed for three weeks and the pain stopped.

October, 1902, he reports continued absence of pain.

Jan. 27, 1903. Ten days ago he began to feel heavy and stupid. Three days ago he began to be a little jaundiced, temperature 100°. He was put on milk and soda for a few days and his symptoms soon disappeared.

Jan. 27, 1904. He has been drowsy for several days, last night he woke up with bad pain and vomiting. He is now a little jaundiced, the stomach contains mucus and bile stained fluid. By January 30 the attack was over.

June 4, a similar attack lasting for a week.

Sept. 14. He has been sick all summer, has lost 35 pounds in weight, pain, jaundice, sometimes fever. Has taken very little food.

He now looks very badly, feeble, emaciated and a little jaundiced.

He was put on three simple meals a day and encouraged to walk. He improved very rapidly.

November 1. He is well and working hard, but takes regular exercise.

CASE X. *Attacks of acute gastritis complicated with a variety of other conditions.* Female, 34 years old, married 10 years, no children.

She is a lawyer by profession, having studied law after her marriage.

She will drink no milk, eats only beefsteak and lamb chops for meat, eats no vegetables except salads with oil, eats sweet desserts, remains in the house for days at a time.

May 30, 1899, an attack of diarrhœa with mucus and loose fecal matter lasting for two weeks.

June 3. She was examined by a gynecologist who reported "Chronic endometritis, position of uterus normal, ovaries not enlarged, curettage recommended."

Jan. 1, 1901. She is having continued menorrhagia, hæmoglobin 55 per cent., feeble and melancholic.

June 21. The menorrhagia having continued without improvement, she was curetted.

Jan. 2, 1902. She has been in better health for the past six months than for many years. Menstruation only lasts for three days, she has gained flesh and strength.

Last night she ate some crab meat. About 1 A. M. she had vomiting and intestinal colic. She was not out of bed until January 4.

Feb. 15, at 6 A. M., she had colic again. She was nauseated all day and at 10 P. M. the pain returned with great violence, most intense in the region below the right breast. She vomited bile-stained fluid. The right leg was drawn up, the whole abdomen was tender and tympanitic, the greatest tenderness was over the region of the gall bladder.

She continued to have abdominal pain, but improved and by April 1 was well again.

June 16. A few days ago she began to have abdominal pain and nausea. This morning the pain is localized in the right iliac region, temperature 99°. At 3 P. M., after an enema, the pain was very severe, temperature 100°. At 9 P. M., she looked very sick, temperature 101°, pulse 100 and small. The abdominal muscles were tense, especially on the right side. A surgeon, in consultation, made the diagnosis of appendicitis with possible general peritonitis and advised no operation.

The next morning the temperature was normal and the other symptoms diminished. In the evening menstruation commenced and she was much better.

Sept. 24, after an indigestible dinner she suffered from indigestion all night and at 1 o'clock in the afternoon had an attack of abdominal pain, vomiting and partial collapse. The next day the abdomen was distended and tender, face pinched, pulse small and rapid, extremities cold. On the 26th she vomited large quantities of green fluid at frequent intervals. After this the bowels moved and she got better.

I saw her first in October, 1902. I succeeded in getting control of her and by regulating the diet and mode of life the gastric symptoms were relieved and her health became good. I heard from her in February, 1906, and she was still well.

CHRONIC CATARRHAL GASTRITIS.

It is customary to include several different lesions of the *stomach* under this name.

Lesions.—1 The only evident change in the stomach is an increased production of mucus. This can be washed out of the stomach during life; it is found adherent to the wall of the stomach after death.

2. Besides the increased production of mucus there is also degeneration of the cells of the peptic glands.

3. There is a growth of connective tissue between the gastric tubules, with deformity and atrophy of the tubules. In some cases the new connective tissue forms little polypoid tumors which project inward.

4. With cardiac disease and with cirrhosis of the liver there may be a well-marked chronic congestion of the mucous membrane.

5. There may be hypertrophy of the muscular and connective coats at the pylorus producing stenosis, and with this there is often dilatation of the stomach.

6. There may be a more diffuse hypertrophy of the muscular and connective-tissue coats with marked diminution in the size of the stomach.

7. The stomach may be considerably dilated without stenosis of the pylorus.

8. There may be considerable exudation of yellow, brown, or green serum into the cavity of the stomach. These belong to the severe and long continued cases of chronic gastritis with new growth of connective tissue and dilatation of the stomach.

Etiology.—A considerable number of cases of chronic gastritis follow acute or subacute gastritis. This is especially true of the gastritis which complicates epidemic influenza, typhoid fever, and so many of the infectious diseases.

Cardiac disease, pulmonary emphysema, and cirrhosis of the liver produce, first, chronic congestion and then chronic gastritis.

Pulmonary phthisis, chronic nephritis, and gout are often accompanied by chronic gastritis.

The habitual use of improper food, of alcohol, and of drugs are ordinary causes.

The same climatic conditions which predispose to chronic nasopharyngeal catarrh and to chronic bronchitis have the like effect as regards chronic gastritis.

Symptoms.—First among the symptoms comes pain, varying from a mere feeling of oppression and discomfort to the most severe and agonizing pain. The pain is in some cases due to the presence of food in the stomach. It comes on at first only with a considerable quantity of food, but later even small fragments of solid food or spoonfuls of liquid food will excite it. Very often patients learn to vomit of their own accord in order to relieve the pain.

In some persons the pain seems to accompany gastric digestion and to cease when this is finished.

In some cases the pain seems to depend on hyperacidity and hypersecretion of the gastric juice.

With alcoholic gastritis there may be a peculiar feeling of soreness in the stomach, which is commonly called heartburn.

Then there are many cases in which we can only say that so long as the patients have gastritis they have pain, and as the gastritis improves so does the pain.

As a rule the pain is at first not constant, but comes on in attacks, which become more frequent and of longer duration as the gastritis progresses.

It is often difficult to distinguish the pain of chronic gastritis from that belonging to ulcer of the stomach, ulcer of the duodenum, biliary calculi, intestinal colic, movable kidney, disease of the uterus, tubes, and ovaries, anæmia, hysteria, and neurasthenia.

Nausea and vomiting are also regular symptoms. The nausea belongs to the early morning hours, but may persist throughout the day. It may show itself as a disgust for food, or as a feeling of anxiety and depression of spirits, or as the actual sensation of impending vomiting.

The vomiting is most frequently a vomiting of food, either in considerable quantities, or vomiting kept up so long as the smallest particle of food is left in the stomach. In some cases of alcoholic gastritis the stomach empties itself about an hour after nearly every meal.

In other cases large quantities of brownish fluid, mixed with mucus and food, accumulate in the stomach and are vomited from time to time. This is especially the case when the stomach is dilated. Regurgitation of an acid fluid before breakfast in the morning is especially common with alcoholic gastritis. In a few cases the patients will vomit nearly every day as much as half a pint of pure mucus.

There are rare cases in which vomiting is kept up as a habit long after the real reasons for it have stopped.

Vomiting of blood may occur in the course of any chronic gastritis, but is much more likely to occur if cirrhosis of the liver or fatty liver, exist at the same time. Usually the hemorrhages are small, but with cirrhosis of the liver large quantities of blood are vomited.

The vomiting may occur in attacks with intervals of days or weeks, or it comes every day. I have known a man to vomit every day for twenty years.

Vomiting is also caused by diseases of the uterus, tubes, and ovaries, by an accumulation of fæces in the colon, by anæmia and by hysteria.

Retention of food in the stomach is especially a feature when there is stenosis of the pylorus or dilation of the stomach, but it is also found with chronic gastritis. A considerable retention of food is regularly attended with vomiting.

Constipation is often present. It seems to depend on a variety of causes and may have nothing to do with the condition of the stomach, but in some cases as the gastritis improves the bowels become regular.

Headache is often a symptom. It follows a variety of types but perhaps the most common is that which comes on at intervals. In some cases it is found that whenever there is headache bile can be washed out of the stomach.

A general loss of health, of which emaciation and loss of muscular strength are the most prominent features, is found with the more severe cases of gastritis. This depends largely upon the gastritis and the interference with digestion, but in some cases it is simply due to starvation. Of their own accord, or by the advice of a physician, they give up one article of food after another until they do not get enough to eat. The exclusive diet of meat and hot water, if kept up for too long a time, has a particularly bad effect.

A considerable number of nervous and hysterical symptoms, and disturbances of sensation in the mouth, throat, and other parts of the body are often present. It is difficult to tell how far these symptoms are to be attributed to the gastritis.

Course of the Disease.—Chronic gastritis seems to have a natural disposition to continue and to grow worse from year to year, but with periods of remission and exacerbation. The histories, therefore, may extend over many years, and the symptoms come on in attacks. Between the attacks the patients seem to be well, but the attacks become more severe, of longer duration, and occur at shorter intervals, as the disease goes on.

I do not know of any satisfactory way of classifying the varieties of chronic gastritis. We can, however, for practical purposes, put the patients together in rather rough groups.

1. The cases in which pain referred to the region of the stomach is the principal symptom.

(a) Pain without vomiting occurs with chronic gastritis by itself, or with retention of food, or with dilatation of the stomach, or with failure of gastric digestion. The patients do not distinguish between these conditions, they only complain of the pain, they often do not think that they have any stomach-disease. The diagnosis has to be made with the stomach-tube.

(b) Pain with vomiting occurs with hyperacidity, with dilatation, and with abnormal sensitiveness of the gastric mucous membrane to the contact of food. The last condition seems to give the most intense gastric pain, a pain that can only be relieved by the removal of the food from the stomach.

2. Nausea and vomiting are ordinary symptoms in all the forms of chronic gastritis, but there are some patients in whom they are particularly prominent. There are persons in whom for many years nausea, with the accompanying depression of spirits in the early morning, is the only symptom for many years. There are persons in whom vomiting, with or without nausea, continues for years, and yet there are no other gastric symptoms.

3. There are patients who for years suffer from attacks of sick-headache, with or without vomiting. Later, instead of the sick-headaches, there are attacks of gastric pain and vomiting.

In some of the cases bile can be washed out of the stomach during every attack, in others no bile is present.

4. There are patients who suffer from retention of food in the stomach, or from dilatation without stenosis in addition to the chronic gastritis. Pain and loss of nutrition are the prominent symptoms. Treatment is often very satisfactory.

5. In the patients with dilatation and stenosis of the pylorus the pain, vomiting, and loss of nutrition get steadily worse. Treatment only relieves the pain and vomiting, the loss of nutrition continues. The question of a surgical operation regularly comes up.

6. There are patients in whom depression of spirits going on to actual melancholia is the most marked symptom. In these cases as the gastritis improves the melancholia disappears. Of course a great many of the cases of melancholia do not have gastritis at all.

7. There are patients in whom the loss of nutrition is out of proportion to the gastric symptoms. They really have a chronic gastritis, but it may be difficult to tell how far this is the cause of the loss of nutrition, and how far it is the thing to be treated. Some of these cases are very difficult to manage.

Treatment.—In attempting to establish a satisfactory treatment for chronic gastritis, it is important to state as clearly as possible the problem which is to be solved.

First, then, we must remember that all the patients who suffer from gastric symptoms do not necessarily have chronic gastritis.

Besides those who have functional disturbance of the stomach, or cancer or ulcer of the stomach, we find many others in whom gastric symptoms are due to diseases of other parts of the body. Anæmia, uterine disease, the neurotic and hysterical condition, and constipation, often behave in this way. In old people the function of gastric digestion is often impaired simply as the result of old age. To each one of these conditions belongs its appropriate treatment, but it is not the treatment of chronic gastritis.

Still further, we must remember that in many cases of gastritis palliation of the symptoms is all that we can hope for. This is true with the gastritis associated with heart-disease, emphysema, phthisis, cirrhosis, Bright's disease, gout, rheumatism, and alcoholism. It is also true of the cases in which the inflammation has gone on to the destruction of the peptic and mucous glands. After excluding all these, there remains a large and important group of cases of chronic catarrhal gastritis, in which we may hope not only to alleviate the symptoms, but to cure the disease.

It is evident, from the nature of the disease, that any treatment intended not merely to palliate, but to cure, must be of long duration, and that it must be repeated from time to time, when the inevitable relapses occur.

The different plans of treatment, then, which may be adopted are:

The curative effects of climate and mode of life.

The regulation of the diet.

The administration of drugs.

The use of local applications directly to the inflamed membrane.

It is unnecessary to lay down rules as to the sort of climate; that can be regulated by the tastes of the patient. The two points of importance are: First, the locality selected must be one where the patient can lead an out-door life. Second, the patient must live in this climate either for several years or for a considerable part of each year.

Excellent as this method of treatment is, it is evident that it can be carried out only by a limited number of persons.

The regulation of the diet is a matter which demands consideration in every case of chronic gastritis. In trying to ascertain the best way of feeding these patients, I have found only one satisfactory method, and that is to feed them experimentally with different articles of food, and then after an interval of several hours wash out the stomach and see how thoroughly these articles of food have been digested and removed from the stomach. After pursuing this course for a number of years I have arrived at the following conclusions:

It is necessary that the patient should be well fed; a starvation diet never answers.

The stomach does not require any rest from the performance of stomach digestion; on the contrary, it is all the better for being called on to perform its natural functions.

The patients' own ideas as to what food agrees with them are usually erroneous. They are apt either to starve themselves or to select the least nutritious articles of food.

The use of artificially digested foods, or of substances, such as pepsin, to assist stomach digestion is unnecessary.

The starches, oatmeal, corn-meal, bread, the cereals, the health foods are, as a rule, bad. Portions of them remain undigested in the stomach for many hours.

Milk in adults is an uncertain article. It answers very well for some persons, not at all for others.

Meat is usually readily and well digested, but there are occasional exceptions to this rule.

Vegetables and fruits can be eaten, but the particular varieties must be selected experimentally for each patient.

I do not believe that any case of chronic gastritis is to be cured by diet alone. Even the exclusive milk diet, while it often relieves symptoms, is, as a rule, only temporary in its effects, so that the patient simply loses a certain amount of time by employing this instead of more efficacious plans of treatment.

The advantageous use of drugs belongs to the earlier stages of chronic gastritis. At that time they often palliate symptoms and sometimes even seem to cure the inflammation. In the later stages of the disease their use becomes more and more unavailing. The reliable drugs for this purpose are not numerous: the preparations of soda, potash, and bismuth, the mineral acids, glycerine, sometimes carbolic acid, sometimes iodoform, sometimes the bitter infusions. If none of these answer, it is hardly worth while to look any further. If we can combine, with the administration of drugs, the regulation of the diet, and of the mode of life of the patient, then, of course, our chances of success are much greater.

THE USE OF LOCAL APPLICATIONS DIRECTLY TO THE GLANDULAR COAT OF THE STOMACH.

This I regard as the most efficacious plan of treatment for those patients who are not able to leave home and seek a proper climate, but ask to be relieved without interruption to their ordinary pursuits. The local applications are readily made by the introduction of a soft rubber tube through the œsophagus into the stomach.

Liquid applications are the best. They should be made in such quantities as to come thoroughly into contact with the entire surface of the mucous membrane, although the pyloric end of the stomach is the region where the inflammation is principally situated. They should be made at a time long enough after eating for the stomach to be as nearly empty as possible.

For many cases warm water alone in considerable quantities is the only local application needed. In some, however, there is an advantage in medicating the water, and for this purpose I employ a variety of substances.

The drugs I use most frequently are chloride of zinc, sulphocarbolate of soda and gomenol.

For the first week it is often necessary to put the patient on a milk diet, and this can be done even with those patients who under ordinary circumstances cannot take milk at all.

Then, after a time, to the milk we add one solid meal composed of meat alone. Next, this single meal is increased by the gradual addition of fruits, vegetables, and bread. Then comes the giving of two solid meals a day, instead of one, then three solid meals, and now we get rid of the milk in part or altogether.

For the first week of this treatment it is wise not to expect any special improvement. Indeed, even a longer time than this may try the perseverance of the physician and the confidence of the patient.

Sooner or later, however, the expected improvement begins; the nausea and vomiting cease, the constipation or diarrhœa is improved, the flatulence is no longer troublesome, the headache becomes less frequent, and, of more real value than these, the improvement in the general condition of the patient becomes evident. The color, the weight, the appetite, the sleep, the spirits of the patient, all show a change for the better. Of all the symptoms, the pain is the one which is apt to persist the longest.

For two or three months the patient has to be kept under observation, and the applications to the stomach made by the physician. After this the patient is dismissed, but continues the treatment himself, first every other day, then twice a week, then once a week for several months. The regular relapses of the disease are managed in the same way, but are much more quickly relieved.

Cases of chronic gastritis with old ulcers of the stomach, with a growth of new connective tissue in the glandular coat, with thickening of the connective-tissue and muscular coats and with stenosis of the pylorus regularly require a gastro-enterostomy to obtain relief.

But to be sure of the diagnosis of these conditions requires time and attention. Pain, vomiting, emaciation, dilatation of the stomach may all exist for months and yet be readily cured without operation.

CHRONIC DILATATION OF THE STOMACH.

For clinical purposes it is convenient to describe three forms of dilatation of the stomach.

1. There is a form of moderate dilatation of the stomach.

without stenosis of the pylorus, without chronic gastritis, without failure of stomach digestion. If not relieved by treatment the condition continues for years, although there are often periods of improvement. The two prominent symptoms are pain in the upper part of the abdominal cavity and loss of nutrition. The pain comes on at intervals, which become shorter and shorter until there is pain every day. The loss of flesh and strength are progressive until the patients can hardly walk.

With these two cardinal symptoms are often associated a variety of accessory symptoms. Some patients complain of dyspnœa, apparently diaphragmatic in character. In some the heart's action is increased in frequency. In women there may be a variety of nervous and hysterical symptoms. The loss of nutrition seems to depend partly on insufficient food, for the patients are apt to leave off one article of food after another with the idea of preventing the pain.

Temporary relief may be obtained by travelling, by an exclusive diet of milk or of meat, by pepsin, bismuth, nux vomica, etc. But the symptoms soon return and grow worse. The only curative treatment is the daily lavage of the stomach. This is very effectual, even in patients who have suffered for a number of years.

2. There is a form of dilatation of the stomach without stenosis of the pylorus, but with chronic gastritis. In these cases the stomach is larger and articles of food are retained in it for a longer time, sometimes for several days. There are pains in the upper part of the abdominal cavity and a loss of flesh and strength which is carried very far. Nausea and vomiting are regularly developed after a time. When the stomach is first washed out, even after twelve hours' abstinence from food, it will be found to contain a large quantity of undigested material.

These patients get well with lavage, but it has to be kept up for a long time.

3. There is a form of dilatation with stenosis of the pylorus. The stenosis is caused by carcinoma, by ulcers, and

by inflammatory thickenings. The dilatations produced in this way are very large and much food is retained. The patients have pain, loss of nutrition, nausea, and vomiting.

The patients are made more comfortable and life is prolonged by lavage, but after a time they do badly, lose flesh and strength and die.

The best thing to do for these patients is a gastro-enterostomy, but even this is not always successful.

Case XI. *Ordinary chronic gastritis; quick recovery.* Female, 23 years old, Nov. 14.

In 1892 she had diphtheria, followed by paralysis, disturbance of heart action and albumen in the urine. From these she recovered entirely. One year ago she began to have pain and discomfort in the epigastrium with headache after meals.

About twice a month there would be a bad day when she would have to stay in bed with hot water bottles.

The pain in the epigastrium and the headache have gradually become worse and of longer duration and the exacerbations more frequent, longer and accompanied with nausea and vomiting.

For the past four weeks she has had pain all the waking hours, nausea every day, vomiting twice a week. The bowels have continued regular, she has not lost weight.

The washings of the stomach showed a considerable quantity of mucus.

After two weeks daily lavage she had no more gastric symptoms and could eat three good meals a day.

Case XII. *Chronic gastritis, the ordinary form.* Female, 24 years old, always well until January, 1881, then she began to have pain after eating which continued until she vomited. These attacks occurred at intervals.

April, 1881, she had scarlet fever, since that time the attacks have occurred once or twice a week.

April 8, 1882. She came to me. The attacks of pain and vomiting every week had continued. She was put under ordinary treatment of stomach washing and regulated diet. By June 13 she was well. I have heard from her from time to time up to 1906 and she has continued to be well.

Case XIII. *Chronic gastritis, pain and vomiting.* Female, 42 years old.

In the winter of 1892-1893 she had several attacks of pain and vomiting, one of them lasted for a week.

From October, 1893, the attacks of pain and vomiting came on every two weeks.

In August, 1894, she began to have diarrhœa. For three weeks before she came to New York she was in bed with constant vomiting and diarrhœa.

Sept. 25, 1894, she came to me. The stomach was washed every day. For two days she was given milk alone, then one meal, then two meals, then three meals.

Oct. 8, the pain and vomiting have stopped, the diarrhœa continued, she was given salol and castor oil.

Oct. 16, she went home apparently well.

She has continued to be well up to 1906.

Case XIV. *Chronic gastritis with severe pain.* Male, 50 years old, dentist.

In the winter of 1867 for the first time he had feelings of oppression after eating which lasted for several weeks. Then he had no more trouble until the winter of 1869. Then the same feeling of oppression after meals came on with greater severity and lasted until the summer. He then went to the country, was better and continued better until 1872.

That autumn he had remittent fever for three weeks and after that intermittent fever for six months.

In 1873, in the winter, there was greater discomfort after eating. After this he was better every summer in the country and worse every winter in New York. Diet and medical treatment were of no service.

In 1878 the winter was very bad. All food was followed by severe pain and he began to try to vomit to stop the pain. The bowels were always constipated.

In July, 1879, he was in the country on a milk diet for five weeks, the pain was relieved and he could again take solid food.

September, 1879, the pain returned worse than ever and milk diet did not relieve it. The pain was severe and nearly continuous so that he hardly slept at all. Vomiting only partly relieved the pain.

November 4, 1879, he came to me. His appearance was that of a man exhausted by suffering and want of sleep. He was living on a little porridge and beef tea. His bowels moved about once a week.

Daily washing of the stomach was begun, he was given porridge in the morning, a chop at noon, milk in the afternoon and beef tea in the evening.

Nov. 10. The pain is not as severe. The daily washings were continued until February, 1880. At that time he had gained 17 pounds, could eat three meals a day without pain and felt well. In December, 1890, he reported himself as having had no further trouble.

CASE XV. *Chronic gastritis with emaciation.* Male, 49 years old.

In July, 1890, he began to lose flesh and strength.

In July, 1891, he began to have abdominal pain, occasional vomiting, headache, loss of appetite and became much weaker and thinner.

Sept. 3, 1891, I saw him first; he complained of the above symptoms and looked thin, pale and sick. I put him on a milk diet.

Oct. 3 he came back, he has felt less pain and has not vomited, but has continued to lose flesh and strength.

The stomach was washed once a day and he was given two solid meals daily for two months. He improved rapidly, regained his flesh and strength and has been well up to December, 1906.

CASE XVI. *Chronic gastritis with exudation of serum, very few gastric symptoms.* Male, 40 years old, April 18, 1903.

For about three years he has suffered from chronic constipation, he takes half an ounce of castor oil every night and an enema in the morning. Within two years he has lost 60 pounds in weight. His appetite is good, he does not vomit, he feels well, keeps at work and sleeps well except when he has pain in the stomach.

The colon is distended with gas, the stomach is not dilated, there is no tumor in the abdomen, the heart and lungs are normal, the urine is normal.

Washing of the stomach showed food and a large quantity of serum.

Until April 27 the stomach contained every day a large quantity of green or brown fluid with little mucus, after that time it washed clear.

May 1 he went home but continued to wash the stomach for three months. He steadily gained in weight and the constipation disappeared.

In December, 1906, he was still well.

CASE XVII. *Headaches with vomiting for many years, apparently due to the presence of bile in the stomach, cured by washing out the stomach.* Female, 48 years old.

When she was sixteen years old she began to have sick headaches.

From her sixteenth to her thirty-second year the headaches occurred at irregular intervals, often brought on by travelling.

The headaches usually lasted twelve hours; during this time she would vomit large quantities of bile and have severe headache, the vomiting seemed to give relief.

From the age or thirty-two to the present time the headaches were no longer brought on by travelling, but continued to occur at irregular intervals.

At the age of forty the headaches began to accompany or follow menstruation, but they also occurred at other times and would last for three days.

From the age of 40 to 45 the stomach could not digest food for several days after an attack. She has gone an entire week without food.

In her forty-seventh year she had the headaches every week.

In her forty-eighth year the headaches came every other week.

May 5, 1891, I saw her first.

She was a large, strong, healthy looking woman, not at all neurotic.

May 6 she felt a headache coming on. Washing the stomach three hours after eight ounces of milk showed no milk, no mucus, but a good deal of yellow bile. The headache and vomiting came on that afternoon and she was in bed until May 9.

May 11 to 16 the stomach was washed every day, always bringing away some bile. She felt all the time as if a headache was impending but yet did not have it.

May 18 to 23 the stomach was washed every day, from the 19th to 21st there was bile in the stomach and the feeling of an impending headache.

June 25, she has washed her stomach every day and has had no headaches. On June 24, in the morning, she felt the approach of a very bad headache. She washed the stomach every hour for eight hours, bringing out large quantities of bile, the headache was averted.

After this she only washed the stomach when she felt a headache was coming on.

CASE XVIII. *Chronic gastritis, displacement of the stomach and right kidney.* Female, 60 years old, began six years ago to have gastric symptoms and has had them ever since. Gastric pain either continuous or in attacks, water brash, sometimes fever and progressive loss of flesh and strength, have been the principal symptoms. She has faithfully tried physicians, cures and diets without relief and is now much discouraged.

March 16, 1905, she came to me. She is a very nice old lady, feeble and emaciated. She complains principally of the water brash and weakness.

The stomach is a little dilated and displaced downward, lavage gives undigested food and mucus. The right kidney is movable.

The case seemed very unfavorable, lavage made her worse, the many attempts at restricted diet have always caused her to lose flesh and strength. However, she remained in New York until April 21 and then reported to me from time to time. The treatment consisted only in the exact regulation of her diet and mode of life. Progress was at first very slow, but by June the water brash stopped and she was fatter and stronger. Since then she has done very well. In October, 1905, she had no gastric symptoms and was leading an ordinary life. She has continued well up to December, 1906.

CASE XIX. *Chronic gastritis, fatty liver, repeated vomiting of blood.* Female, 49 years old, Nov. 13.

The woman was habitually intemperate. Fifteen months ago she fell and struck her abdomen against the edge of a box. She had some pain in the abdomen and vomited blood.

Two months ago she became jaundiced and vomited blood. Since that time she has lost flesh, has had pain in the epigastrium and has vomited blood every day. She has also noticed an enlargement of the liver.

Now, she is fat, jaundiced, weak, has pain in the abdomen and vomits blood. The liver is very large.

She continued to vomit blood and mucus every day until she died on December 29.

Autopsy. The body was deeply jaundiced.

The heart and lungs were normal.

The mucous membrane of the stomach and small intestine was thickly coated with mucus.

The liver weighed 6 pounds, the cells were infiltrated with fat.

CASE XX. *Chronic gastritis with thickening of the wall of the stomach.* Male, 56 years old.

Five months before his death he began to have pain, nausea and vomiting after eating. These symptoms continued steadily with increasing emaciation until he died. It was a hospital case badly observed and badly taken care of.

Autopsy. The brain and heart were normal. In the lower lobe of the right lung there was an area of gangrene about three inches in diameter. There was old fibrous induration at the left apex.

The capsule of the liver was thickened and studded with minute white nodules.

The spleen and kidneys were normal.

The wall of the stomach was everywhere thickened, the mucous membrane projecting inwards in flat nodules, the pyloric opening nearly closed. There is a growth of round cells and connective tissue in the glandular coat with some obliteration of the tubules. The flat nodules are made of reticulated connective tissue. The connective-tissue and muscular coats are much thickened.

The peritoneal cavity contains turbid serum and the entire p ritoneum is studded with minute white nodules.

CASE XXI. *Temporary dilatation of the stomach; recovery.* Male, 37 years old, came to me in August, 1901.

Fifteen months ago, after indiscretion in eating, he had a bad attack of vomiting and diarrhœa, after that there was no nausea, vomiting or gastric pain, but he lost 20 pounds in weight and was unable to work. He put himself under the care of a stomach specialist, was fed eight times a day and took a good deal of medicine without improvement.

When he came to me the stomach was much dilated with some retention of food.

I treated him off and on for five months with lavage and medicines, without any improvement.

Then I stopped all treatment, gave him one solid meal a day with milk the rest of the time. He was decidedly better in a week. At the end of two months he had gained flesh and strength and the stomach had returned to its normal size. After this he took two meals a day.

January, 1903, he had continued to be very well and the stomach was of normal size.

CASE XXII. *Severe and protracted chronic gastritis with dilatation of the stomach; recovery.* Female, 20 years old, March 19, 1903.

For several years she has been having trouble with her stomach on and off. At times the gastric symptoms have been associated with menstrual disturbances, repeated examinations by gynecologists have failed to reveal any pelvic lesion. For the past year she has not menstruated at all. Pain, nausea, vomiting and loss of strength have alternated with periods of improvement. For the past year the vomiting has stopped, but the pain and the loss of flesh and strength have continued.

During all this time she has been treated by a variety of physicians and has travelled abroad. There have been periods of improvement, but she has continued to get worse.

She now looks very badly. She is feeble, emaciated and anæmic, menstruation has stopped, she takes hardly any food on account of the abdominal pain. The stomach is dilated down to the umbilicus. The washings of the stomach contain a large quantity of green serum and a little food.

The case looked altogether as if it could only be relieved by gastro-enterostomy.

March 26 I began with daily lavage and the attempt to feed her. For some time milk, cereals, bread and eggs would remain undigested in the stomach and she had to be fed principally on meat.

April 2. The stomach washes clean, she is a little better.

April 20, she has improved, the stomach washes clean except for a little mucus, she can eat a greater variety of food, she has gained a little flesh and can walk a short distance.

May 15, she still improves, but has bad days and is not strong.

After this the lavage was continued every day, she could eat better and improvement was more rapid.

October 14. She now has gained 20 pounds in weight, feels strong and well and has no gastric symptoms. The stomach has returned to its natural size, the lavage is to be used less frequently.

Feb. 23, 1904. She is fat and well, menstruation is now regular, she has stopped the lavage, can eat well and has no gastric symptoms.

January, 1907. She continues to be very well.

CASE XXIII. *Chronic gastritis, dilatation of the stomach; tacchycardia; recovery.* Male, 29 years old, Nov. 14, 1902.

A strong, active, well-built engineer was well until eight months ago. Then he had a sudden attack of dyspnœa, pain in the epigastrium, and nausea. Since that time he has had many attacks of dyspnœa, pain in the epigastrium, nausea, trembling and sweating, he has only vomited twice, he has lost 40 pounds in weight.

Now, the lungs and heart are normal, except that the pulse is 100. The stomach extends from the left axillary region down to the navel, hæmoglobin 70 per cent.

He was ordered to take only two full meals a day, morning and evening, and to wash out the stomach before the evening meal.

Dec. 12, he reports improvement. The pain in the epigastrium is almost gone, he can sleep at night, he has gained two pounds, the dyspnœa is better, the stomach washes clean, it is now of normal size.

He was ordered to continue the same treatment, but to take also bread and milk at noon.

Dec. 26, he was doing well until five days ago. Since then he has had bad pain in the epigastrium, dyspnœa, pulse 120, has lost two pounds, heart action regular, no murmur, stomach contains food and mucus.

Ordered to stop work, stop lavage, to take sulpho-carbolate of soda and belladonna.

Jan. 2, 1903, he has no pain, pulse 100, feels better, to continue.

Jan. 16, he has gained 25 pounds, the stomach extends from the fifth interspace to one inch above the navel, pulse 100, he still has pain in the epigastrium.

Jan. 22, the stomach is smaller, pulse 100.

Feb. 2, he has had two bad attacks of pain and palpitation, pulse 110, no dilatation of the stomach, to take digitalis, nux vomica and belladonna instead of the previous medicine.

Feb. 9, no better, another attack of dyspnœa, pulse 90, change to sodium bromide and nux vomica.

March 21, he has no pain or dyspnœa, has been working, stomach is not dilated, pulse 125.

April 17, no pain, no dyspnœa, he has been working, pulse 130.

May 16, pulse 120, he has continued to work, some pain in the epigastrium, to take asafœtida instead of sodium bromide.

May 28, pulse 78, he has lost 4 pounds, the stomach is again dilated.

Oct. 10, he has gained weight, has been working steadily, pulse 100, the stomach is not dilated, he has irregular pains.

Dec. 8, pulse 96, is gaining in weight, has no pain and feels well, to stop the asafœtida.

After this he did well without treatment.

April 16, 1904, he weighs 190 pounds, pulse 70, stomach is not dilated, he seems to be entirely well.

CASE XXIV. *Chronic gastritis, stenosis of the pylorus, dilatation of the stomach, gastro-enterostomy; death.* Male, 43 years old, Sept. 14, 1894.

About one year ago he began to lose flesh and strength, lost appetite and vomited occasionally.

In August, 1894, the vomiting was more frequent and the quantities very large, there was pain in the upper part of the abdomen, he became very feeble and emaciated.

Sept. 14, the man looks very badly, the largely dilated stomach is easily made out, but no tumor can be felt. The stomach washings show large quantities of food and exudate. He was made comfortable by the daily washing of the stomach but continued to lose flesh and strength.

Nov. 28, a gastro-enterostomy was made by Dr. McBurney. The man seemed to do well for a week and then quietly died.

Autopsy. The intestinal anastomosis was perfectly tight, there was a little fibrin on the peritoneum.

The stomach was very much dilated. The pyloric end is thickened and stenosed by an hypertrophy of the connective-tissue and muscular coats. Just above the pylorus over a space an inch and a half in diameter the glandular coat has been destroyed. Through the rest of the wall of the stomach there is a growth of connective tissue with atrophy and deformity of the tubules.

CASE XXV. *Chronic gastritis, stenosis of the pylorus, dilatation of the stomach.* Male, 50 years old.

In December, 1875, he stated, that for eighteen months he had suffered from pain and vomiting after eating. The pain in the epigastrium came on ten minutes after eating, the vomiting half an hour later. He vomited from one to three times a day.

He was taught how to wash out the stomach once a day, the pain and vomiting stopped and he did very well until February, 1877. Then the pain and vomiting returned and continued, the vomited food was mixed with coffee-ground matter, no tumor could be felt, he steadily lost flesh and strength until April 24, when he died.

Autopsy. In the heart the aortic valves were a little thickened.

Both lower lobes of the lungs were congested and œdematous.

The liver and spleen were normal.

The kidneys were of medium size, chronic nephritis.

The stomach was dilated and contained blood. All the coats of the stomach were thickened. At the pylorus the thickening was so great as to nearly close the opening. There is a thick layer of new tissue beneath the mucous membrane, partly replacing it and also infiltrating the thickened muscular coat. This new tissue is composed of a reticulated stroma and small round cells.

CASE XXVI. *Tubercular gastritis.* Male, 36 years old; was sick for about five years with ordinary pain and vomiting of a chronic gastritis. The symptoms were at first relieved by treatment, but returned over and over again until he died in a condition of extreme emaciation.

Autopsy. The stomach was very small, the pyloric orifice was nearly occluded. The wall of the stomach was thickened, especially around the pylorus. The glandular coat of the stomach was almost entirely destroyed. The muscular coat was thickened.

The principle thickening was of the connective-tissue coat. This was effected by a growth of dense connective tissue in which were scattered little aggregations of round cells and well marked tubercle granula. In the granula there were tubercle bacilli.

CASE XXVII. *Croupous gastritis.* Male, 47 years old.

He stated that he had been well until eight days before his death. Then he caught cold and three days later again caught cold.

Two days before death he was first seen. Then his temperature was 102°, pulse 120, face flushed, pain and tenderness over the abdomen. He looked very ill, but there were no more definite symptoms until he died.

Autopsy. Two-thirds of the inner surface of the stomach, involving the lesser curvature and the anterior and posterior walls, extending nearly to pylorus and œsophagus, is covered with a thick adherent false membrane.

This membrane is composed of fibrin and pus cells. The gastric tubules are not changed. The connective-tissue coat is infiltrated with fibrin and pus.

The rest of the body was examined and no lesions were found.

SUPPURATIVE GASTRITIS.

Lesions.—There is a suppurative inflammation beginning in the connective-tissue coat of the stomach, and extending to the other coats. This inflammation may be circumscribed, with the formation of an abscess; or diffuse, with a purulent infiltration of the entire connective-tissue coat. The abscesses may rupture into the cavity of the stomach. The inflammation of the peritoneal coat may give rise to a general peritonitis.

Causes.—The disease belongs to adult life. It is more common in males than in females. There is, in some cases, a history of the over-eating of indigestible food. It evidently belongs to the class of infectious inflammations.

Symptoms.—The formation of the abscesses in the wall of the stomach is attended with pain in the epigastric region, vomiting, a febrile movement, and the formation of a tumor. If the abscess ruptures into the stomach, the pus may be vomited. The peritonitis often remains localized. The disease may run an acute or a chronic course.

The diffuse suppurative inflammation of the wall of the stomach runs an acute course, terminating fatally in from three to eighteen days. The patient is suddenly attacked with vomiting, pain, and tenderness over the stomach, fever, and great prostration. The symptoms of general peritonitis are soon added, and the patients rapidly get worse.

Treatment.—The best that we can do seems to be to alleviate the patient's symptoms by the liberal use of opium.

Ulcer of the Stomach.

Lesions.—Ulcers of the stomach are usually single, but two or more ulcers may be formed at the same time, or successively.

Welch, from a collection of 793 cases, gives the position of such ulcers as follows:

On the lesser curvature, 288.
On the posterior wall, 235.
At the pylorus, 95.
On the anterior wall, 69.
At the œsophageal end, 50.
At the fundus, 29.
On the greater curvature, 27.

The ordinary diameter of these ulcers is from half an inch to two inches, but some are very small, and some are much larger. Two or more ulcers may become joined, and so form a large ulcerated area.

The ulcers are round or oval, largest in the glandular coat. They may destroy only the glandular coat, or the entire thickness of the wall of the stomach. The edges of the ulcers are clean-cut, and their floors smooth; but sometimes the edges are much thickened, and the floor may be formed by tissues which have become adherent. The edges and the floor are formed of amorphous granular matter, or of connective tissue.

If the patients recover, the ulcers cicatrize either with or without deformity of the stomach.

If the ulcers perforate the wall of the stomach, this perforation may be large and sudden, with the escape of the contents of the stomach into the abdominal cavity; or the perforation may be small, and set up a localized peritonitis; or the opening may be entirely closed by adhesions.

The ulcer may erode either the arteries or the veins of the stomach. With an ulcer there is more or less chronic gastritis.

Causes.—These ulcers are said to be twice as common in women as in men. They have been observed at nearly every age, but seven-tenths of them are in people between twenty and forty years of age.

The ordinary explanation of the way in which these ulcers are formed is, that by embolism, thrombosis, or chronic endarteritis one of the branches of the gastric artery is occluded. The corresponding portion of the wall of the stomach dies and is destroyed by the action of the gastric juice.

Symptoms.—Of all the symptoms pain is the most constant, it is absent only in exceptional cases, it varies in its quality, its intensity, its situation, and its duration.

The most characteristic pain is severe paroxysmal pain localized in a circumscribed spot in the epigastrium, coming on soon after eating and disappearing when the stomach is emptied. Of more common occurrence are paroxysms of severe pain diffused over the epigastrium and radiating in different directions. In many cases both these forms of pain exist: In the intervals between the paroxysms of pain there is often a more or less constant dull pain or feeling of discomfort at the epigastrium. The position of the pain is usually at or a little below the ensiform cartilage. It may, however, be felt as low as the umbilicus, or on either side in the hypochondriac regions.

The pain usually comes on within half an hour of taking food and continues until the stomach is emptied by vomiting or by the passage of food into the duodenum.

The pain is regularly increased by pressure over the stomach, by fatigue, and by exposure. It is diminished by rest and the recumbent position. The pain may recur at regular intervals, it may stop altogether for days or weeks.

Besides the pain belonging to the gastric ulcer the patients may also have pain due to chronic gastritis, to hyperacidity, to retention of food, to dilatation of the stomach, or to localized peritonitis.

Next to pain, vomiting is the most frequent symptom of gastric ulcer. It is apt to occur soon after taking food, or after an attack of gastric pain. The patients may only vomit occasionally, or every day, or very frequently. In some cases the irritability of the stomach is so great that no food can be retained, and the effort at vomiting will be made even when the stomach is entirely empty. The vomiting seems to be due partly to the ulcer, partly to the accompanying chronic gastritis.

It is estimated that vomiting of blood occurs in about one-third of the cases of gastric ulcer. This estimation is not of much value, on account of the occurrence of vomiting of blood with chronic gastritis and simple anæmia.

The bleeding may be preceded by pain, vomiting, and disturbances of digestion, or it may come on suddenly without any other gastric symptoms. The quantity of blood lost may be small, or larger; the larger hemorrhages are those which are most characteristic of gastric ulcer. The most important bleedings are those which are due to the erosion of a blood-vessel in the floor of the ulcer. The blood may be entirely vomited, or some of it may pass into the intestine and be discharged with the stools. With small bleedings all the blood may pass into the intestine, none of it being vomited. Rarely the patients die suddenly without any vomiting, and at the autopsy the stomach is found to be full of blood.

It is not generally understood that patients with either pernicious or simple anæmia may vomit large quantities of blood, and even bleed to death without any ulcer or erosion of the stomach. Such large bleedings, due to anæmia, and not to ulcer of the stomach, are of frequent occurrence and yet are recognized by few physicians.

The importance of a correct diagnosis is very great. If the bleeding is due to anæmia and the patients are treated for ulcer of the stomach they are very apt to die. If, on the other hand, it is appreciated that they have no stomach lesion, and they are treated for anæmia they usually recover.

It is to be remembered in this connection that many anæmic patients have pain after eating, nausea, and vomiting without any disease of the stomach.

Any of the symptoms which have been described under the heads of functional disorders of the stomach and chronic gastritis may occur in cases of gastric ulcer.

The frequency of perforation of the ulcers is variously stated by different authors. Welch gives it as six and one-half per cent. of all cases of gastric ulcer.

It is said that perforation occurs two or three times oftener in the female than in the male, and that in the female the liability is greatest between fourteen and thirty years of age. In the male there seems to be no greater liability to perforation at one age than at another. Ulcers of the anterior wall of the stomach perforate more frequently than those in other situations. The perforation may be preceded by the characteristic pain and vomiting of gastric ulcer, or only by indefinite gastric symptoms, or it may occur suddenly in a person who is apparently perfectly well.

If the perforation is such that the contents of the stomach escape into the peritoneal cavity there is at once a severe pain and the patients pass into the condition of collapse. If they survive the shock of the perforation an acute general peritonitis is developed which is regularly fatal.

The perforation may be small and limited by adhesions. Then a localized peritonitis with collections of pus is set up around the perforation. In this way are formed the subphrenic abscesses which perforate the diaphragm and simulate empyema or pyo-pneumothorax.

The Course of the Disease.—There may be no symptoms during life, and the ulcer, or its cicatrix, is found after death from some other disease.

There are cases which last for weeks, months, or years. The symptoms are marked, but more or less severe. Some of the patients recover, others die of starvation.

There are cases in which one or more large hemorrhages form the prominent, and sometimes the only, symptom.

There are cases in which the large or small perforation of the wall of the stomach forms the prominent feature.

There are cases in which the deformity of the stomach, produced by the cicatrization of the ulcer, gives symptoms for the rest of the patient's life.

Treatment. Ulcer of the stomach is at the best a serious lesion. At the present time the points to be remembered are:

1. Pain, nausea, vomiting and vomiting of blood in anæmic girls are not the symptoms of ulcer of the stomach, but of anæmia and are to be treated accordingly.

2. Medical treatment for ulcer of the stomach is really treatment for the hyperacidity, the ulcer runs its course in its own way. The best plan of management seems to be that of Lenhartz. The patients are kept in bed for four weeks. An ice bag is kept continuously over the stomach for two weeks. On the first day, even after bleeding, the patient receives 200 cubic centimetres of iced milk in spoonfuls and from two to four beaten eggs. The quantity of milk and eggs is gradually increased, but not more than one litre of milk is given at any time. On the sixth day scraped beef is added.

3. Pain and vomiting, even if continued for months, do not necessarily mean an ulcer of the stomach.

4. The so-called exploratory operations do not always establish the diagnosis. The best surgeons spend very little time in looking for the ulcer, they make a gastro-enterostomy and close the wound as soon as possible.

5. If the diagnosis of ulcer of the stomach is reasonably probable the best thing for the patient is a gastro-enterostomy.

CASE XXVIII. *Ulcer of the stomach; death.* Female, 27 years old, Feb. 15.

Seventeen months ago she began to vomit blood in the form of dark, coffee-ground matter. The vomiting was more severe after taking food. There was severe pain in the epigastrium, made worse by food.

Now, she is emaciated, frequent vomiting and severe pain, pulse and temperature normal.

In spite of all treatment the pain, vomiting of food and coffee-ground matter and emaciation continued until she died on April 21.

Autopsy. The brain was not examined.

The heart, liver and kidneys were small, but not diseased.

The lungs were normal.

In the stomach just above the pylorus on the lesser curvature was an ulcer of which the edges had been drawn together so as to form a slit at right angles to the long axis of the stomach. By this contraction the stomach was drawn into an hour-glass shape. The mucous membrane was pale and coated with mucus.

CASE XXIX. *Ulcer of the stomach.* Female, 31 years old, May 14, 1877.

September, 1874, she began to have sour stomach, pain after eating relieved by vomiting, and vomiting of coffee-ground matter.

The attack lasted for eighteen months and then she was better.

Six weeks ago she began to have pain in the epigastrium, constant vomiting, and passed blood from the bowels.

She continued with the pain and vomiting until she died, August 6, 1877.

Autopsy. The stomach was of normal size, its mucous membrane was congested. At the middle of the lesser curvature there was a small ulcer partly cicatrized and puckering the walls of the stomach.

CASE XXX. *Multiple ulcers of the stomach.* Male, 65 years old, May 23, 1885.

Three months ago he had pneumonia. Six weeks ago he began to have chills every morning, not followed by fever.

Now, he is emaciated, tongue coated, pulse 68, temperature 95°, breathing 46, May 26 he died.

Autopsy. The left pleural cavity was half full of serum, the pleura was coated with fibrin. There are old miliary tubercles in the lungs. The right lung is covered with adhesions and contains a few old miliary tubercles.

The pericardium is adherent, thickened and coated with fibrin. In the left ventricle there is a small ante-mortem thrombus.

The liver, spleen and kidneys are normal. The stomach is small, it is coated with mucus. At the pyloric end there is some thickening of all the coats. On the lesser curvature three inches above the pylorus is an ulcer half an inch in diameter, apparently cicatrizing. Half an inch above this is a second ulcer, completely cicatrized. Besides these there are six small ulcers scattered about the stomach with sloughs adhering. The entire glandular coat shows a growth of connective-tissue with atrophy of the tubules.

CASE XXXI. *Ulcer of the stomach and duodenum, misleading history.* Female, 32 years old, March 17.

She said that she had been well until two months ago. Then she began to have a cough with muco-purulent sputum and to lose flesh and strength, no hæmoptyses or fever.

Three weeks ago she had a miscarriage at the fifth month. The placenta was retained for three days and she has not been well since. A tumor can be felt through the vagina in the right broad ligament.

She died April 8.

Autopsy. The brain was not examined.

The heart was normal.

There was muco-pus in the bronchi and fibrin on the pulmonary pleura.

In the stomach there was a perforating ulcer in the anterior wall near the pylorus. In the duodenum there was a small, perforating ulcer near the pylorus. Around these ulcers is a large purulent cavity and all the surrounding tissues are infiltrated with pus. The spleen is involved in the suppurating cavity and is partly broken down.

There is an abscess below the right broad ligament of the uterus.

CASE XXXII. *Perforating ulcer of the stomach.*

Three years ago she began to vomit after eating and continued to do so for three months, then the vomiting stopped.

One year ago she again began to vomit and continued to do so for four months.

Since then there have been no vomiting or gastric symptoms until two months ago.

Then she again began to vomit, the vomited matters were sour and sometimes contained blood.

Now, she is emaciated, has a good appetite, has constant pain over the stomach and desires to vomit. She gradually became more feeble and died.

Autopsy. There was fibrous induration at the apices of both lungs.

The heart was normal.

On the lesser curvature of the stomach near the pylorus was an opening which admitted four fingers, the opening leads into a sac the size of a goose's egg, the floor of which is formed by the pancreas. Behind the pancreas an opening in the sac is closed by the liver.

Case XXXIII. *Ulcer of the stomach; death from bleeding.* Male, 42 years old, Sept. 6.

No previous history.

Sept. 2 he vomited blood and became very weak.

Sept. 3 he again vomited blood.

Sept. 6 he vomited a large quantity of dark fluid and clotted blood.

Sept. 7 he died.

Autopsy. The brain was anæmic.

The lungs were emphysematous, there were small, recent, red blood clots adherent to the pleura.

The heart was normal.

The liver was covered with old adhesions.

The spleen and kidneys were normal.

The stomach was distended with dark, fluid blood. On the lesser curvature close to the pylorus was an ulcer one inch in diameter.

In the duodenum close to the pylorus was a small ulcer.

The intestines contain black blood.

Case XXXIV. *Ulcer of the stomach from an embolus.* Female, 34 years old, Sept. 30.

Two years ago she had attacks of vomiting and headache.

Two months ago she became too weak to work.

Three weeks ago, in the night, she had a chill followed by general pains, vomiting and diarrhœa. Since then she has been in bed and vomits everything.

October 9 she died.

Autopsy. The brain was not examined.

The heart was normal.

In the lungs at both apices there were small areas of cheesy nodules and fibrous induration.

The liver was large and fatty.

In the abdomen on the left side is a sac shut in by the broad ligament of the liver, adhesions between the left lobe of the liver and the diaphragm, and adhesions between the greater curve of the stomach and the abdominal wall. This sac contains gas, blackish fluid and food.

On the greater curvature of the stomach near the pylorus is an ulcer one inch in diameter. Its edges are ragged, gangrenous and coated with fibrin. In the ulcer is a gangrenous piece of the wall of the stomach still adherent. In the gastro-epiploic artery is a plug one-eighth of an inch long.

There is a little fibrin on the peritoneum.

CANCER OF THE STOMACH.

Lesions.—The new growth follows the anatomical types of colloid cancer, or cancer with cylindrical epithelial cells, or of cancer with small polygonal cells. The growth seems to originate in the glandular coat.

The most common shape for the new growth to take is that of a flattened tumor with necrotic and ulcerating centre, which projects inward into the cavity of the stomach. Instead of this, however, the tumor may be quite large and of polypoid shape. In still other cases there is no tumor, but a flat infiltration which occupies more or less of the wall of the stomach. There is usually more or less chronic catarrhal inflammation of the glandular coat. The peritoneal coat is often thickened and adherent to the surrounding viscera. The process of ulceration may involve not only the new growth, but also the wall of the stomach, and even extend into the adherent viscera.

While the great majority of cancers of the stomach are primary, yet secondary tumors have also been observed. Welch has collected 37 such cases, 17 secondary to cancer of the breast, 8 to cancer of the œsophagus, 3 to cancer of the mouth or nose, and the remainder to cancer of other parts of the body.

Cancer of the stomach is very often attended with the growth of metastatic tumors in other parts of the body. Welch gives the following table, based on 1.574 cases :-

Lymphatic Glands.	Liver.	Peritoneum Omentum Intestine.	Pancreas.	Pleura and Lung.	Spleen.	Brain.	Other parts of the body.
551 35 p. c.	475 30.2 p. c.	357 22.7 p. c.	122 7.8 p. c.	98 6.2 p. c.	26 1.7 p. c.	9 0.6 p. c.	92 5.8 p. c.

Causes.—The disease is equally common in males and females. The maximum liability to the disease is between the ages of 60 and 70 years, but gastric cancer is common enough after the age of forty, and is even occasionally seen in persons not over 20 years old.

Symptoms.—The appetite as a rule is poor, either because the ingestion of food causes pain or nausea, or because there is actual distaste for food. But there are exceptional cases in which the appetite continues to be very good.

Nausea and vomiting are often present. The vomiting is of food, of brownish or yellow fluid, of coffee-ground matter, or of blood. The vomited matters may have an offensive odor. This foul smell is more constant in the contents of the stomach when they are washed out than when they are vomited. When the stomach is dilated from stenosis of the pylorus the quantity of vomit at one time is often very large. The vomiting may be an early symptom and continue throughout the disease. Or it may not come on until the disease is far advanced. Or the vomiting may accompany the early stages of the growth and then stop, not to begin again. In a moderate number of cases there is no vomiting at any time; this may be the case even when there is stenosis of the pyloris.

It is said that the situation of the cancer exerts great influence upon the frequency of vomiting, and the time of its occurrence after meals; that when the cancer involves the pyloric orifice, vomiting is rarely absent, and generally occurs an hour or more after a meal. As the stomach becomes dilated the vomiting comes on longer after a meal, sometimes not until after one or more days. Next to pyloric cancer it is cancer involving the cardiac orifice which is most frequently accompanied by vomiting. Here the vomiting occurs immediately after taking food. If there is stenosis of the cardiac orifice the food is regurgitated. When the cancer is situated in other parts of the stomach and does not obstruct the orifices, vomiting is more frequently absent. There are a great many exceptions to these rules, so many that the rules themselves are not of much practical importance.

Bleeding from cancers of the stomach is usually in small quantities, and the blood remains in the stomach long enough to be changed into coffee-ground matter, but sometimes there are very large hemorrhages.

The absence of free hydrochloric acid and the presence of lactic acid in the stomach are of some assistance in making a diagnosis.

Pain referred to the region of the stomach is a frequent symptom and the pain may be very severe, but the number of patients who do not at any time have pain is considerable.

The presence of a tumor is the most certain evidence of gastric cancer. We look for tumors of the pylorus in the epigastric region, unless the stomach is dilated, then the tumors are found lower down in the abdomen. The tumors of the greater curvature and of the anterior wall correspond in their situation to these parts of the stomach. All these tumors are most easily felt if the patient takes a deep breath so as to depress the diaphragm. The tumors usually move pretty freely up and down with the movements of the diaphragm, but sometimes they are fastened down by adhesions. If they are close to the abdominal aorta the tumors seem to pulsate. As a rule there is no great difficulty in making out that the tumor is separate from the aorta, but sometimes it is exceedingly difficult to distinguish between cancer of the stomach and aneurism of the aorta.

Cancers of the cardiac end of the stomach cannot be felt.

The ease with which the tumor can be felt depends not only on its position, but also on its size and shape. Some of the flat infiltrations of the wall of the stomach never make an appreciable tumor. The flattened and polypoid tumors cannot be felt until they have reached some size, so that often enough we do not make out the tumor until a few months before death. If the lympathic glands are adherent and infiltrated they increase the size of the tumor very considerably.

It is not uncommon for persons to have a chronic gastritis with its attendant symptoms for a number of years before the cancer is formed. The symptoms of the gastritis and those of the cancer then seem to make one continuous history.

In the later stages of the disease there may be a moderate rise of temperature. The bowels are usually constipated, but there may be diarrhœa. When there is bleeding from the stomach some of the blood may come away with the stools.

The loss of flesh and of strength are sometimes the first symptoms to attract attention, but more frequently they come after the gastric symptoms have already existed for some time. A very considerable loss of flesh accompanying gastric symptoms always makes one think of cancer of the stomach. But it must not be forgotten that there are exceptional cases in which there is no emaciation and the general health continues to be remarkably good.

There is regularly some diminution in the quantity of hæmoglobin and the number of red blood-cells, the color of the skin changing to white or yellow. In some cases, however, these changes in the blood are as marked as they are in pernicious anæmia, and when no tumor is present the diagnosis becomes very difficult.

Late in the disease there may be œdema of the legs, ascites, and thrombosis of some of the veins, especially those of the legs.

A coma with dyspnœa, like that of diabetes, has been observed in a few cases of gastric cancer.

The ordinary duration of the disease is about one year, but some cases go on much longer—for two, or even three years.

COURSE OF THE DISEASE.

1. A considerable number of cases give a characteristic history—first the gastric symptoms, then the loss of flesh and strength with a tumor in the upper part of the abdomen evidently connected with the stomach.

2. Not infrequently we see patients with well-marked gastric symptoms and some loss of flesh and strength, but there is no tumor, and the diagnosis may remain doubtful for some time.

3. There are patients in whom the gradual loss of nutrition and cachectic conditions are such as to make one feel sure there is a cancer somewhere in the body, but there are no local symptoms and no tumor to tell where it is.

4. There are patients who up to the time of death have the symptoms and the condition of the blood which belong to pernicious anæmia, with very little to call attention to the condition of the stomach.

5. There are cases with a primary cancer of the stomach, which is of small size and gives but few symptoms, and large secondary tumors in the liver. These patients behave as if they had cancer of the liver rather than cancer of the stomach.

6. A primary cancer of the stomach may be followed by the formation of secondary tumors in the peritoneum, with fluid in the peritoneal cavity. In some of these cases the symptoms of the cancer of the peritoneum are much more marked than those of the gastric cancer.

Treatment.—The patients can often be made much more comfortable, and even enabled to eat ordinary meals, by the daily washing out of the stomach. Another plan is to feed them every two hours with small quantities of easily digestible food. If the pain is bad, morphine has to be used.

The question of operation is hardly settled. Some surgeons are enthusiastic as to the good effects of an early operation. Certainly in some cases pain and vomiting can be relieved by a gastro-enterostomy.

CASE XXXV. *Cancer of the stomach, large growth.* Male, 64 years old, Nov. 12.

He says that he was well until one year ago. Then he began to have pain in the epigastrium and nausea after eating. These symptoms were not severe for six months. Then the pain became worse with frequent vomiting.

He is now much emaciated, with a tumor in the epigastrium. He continued to vomit, the tumor increased rapidly in size, he died Jan. 4.

Autopsy. There were no lesions except of the stomach and liver.

There was a mass, larger than a man's fist, situated at the smaller curvature of the stomach, consisting partly of enlarged glands, partly of the new growth in the stomach. The wall of the stomach over its lesser curvature from the pylorus nearly to the cardia is infiltrated with the new growth, which projects into the cavity of the stomach in fungous masses. For about three inches from the pylorus the entire circumference of the stomach is infiltrated with the new growth. The growth consists of large polygonal, nucleated cells. The cells are so close together that no definite arrangement can be made out.

The pancreas is not involved. The liver contains several small nodules of new growth.

CASE XXXVI. *Cancer of the stomach, circumscribed tumor.* Male, 50 years old, November 12.

One year and eight months ago he fell fifteen feet and struck his abdomen. Since then he has had continual gnawing pain in the epigastrium and flatulence.

Eight months ago he began to vomit about two hours after each meal and lost flesh and strength. After a time the vomiting stopped.

Two months ago he began to vomit again, but still had a good appetite.

Now, he is pale and feeble, his appetite is good, he vomits occasionally, has pain in the epigastrium, diarrhœa and œdema of the legs. The urine is normal. No tumor can be felt. He continued in this condition through December, January and February, sometimes vomiting, eating fairly, always with loose bowels.

March 25 the diarrhœa stopped.

April 28 he had a pneumonia involving the lower lobe of the left lung, which resolved by May 19.

He went along in this way, getting weaker, sometimes vomiting, sometimes with diarrhœa, no tumor could be felt at any time. He finally died in November, a year after he entered the hospital.

Autopsy. The brain was not examined.

The heart was normal.

The lungs were emphysematous.

The liver was slightly enlarged, no tumors.

The pancreas was small and hard.

The small and large intestine were coated with mucus.

The kidneys were a little enlarged.

The spleen was large and firm.

The stomach was filled with food.

On the lesser curvature and posterior wall four inches above the pylorus was an ovoid tumor as large as a turkey's egg, attached by a broad pedicle to the wall of the stomach. The new growth was lobulated and of almost villous appearance. It was made up of large, tubular follicles lined with cylindrical epithelium.

CASE XXXVII. *Cancer of the stomach, general dropsy.* Male, 58 years old, Dec. 12.

When he was twenty-nine years old he had a chancre, he was always intemperate. Six months ago his feet and eyelids became œdematous, followed in a week by ascites.

Since then he has had cough and dyspnœa, pain and flatulence after eating, occasional vomiting after coughing, urine diminished in quantity.

Now, he is fairly nourished, pale, no appetite, pulse and temperature normal, urine 45 ounces, trace of albumen, systolic murmur at the apex of the heart, subcrepitant rales over the lower lobes of both lungs, fluid in the right pleural cavity, both legs œdematous, ascites. The liver is small, displaced downward and floating in the fluid so that it could be mistaken for a large liver.

He went on with general dropsy and occasional vomiting until he died on February 20.

Autopsy. The brain was not examined.

The large arteries were atheromatous.

The right ventricle of the heart was hypertrophied and dilated, the left ventricle was much dilated, the aortic and mitral valves were thickened and insufficient.

There was fluid in both pleural cavities, the lungs were emphysematous with chronic bronchitis.

The liver weighs three pounds, it is displaced downward, floating in the fluid which fills the abdomen. It is cirrhotic and contains small white nodules of new growth.

In the stomach, close to the pylorus, on the lesser curvature, is a large, flat ulcerating tumor.

The new growth in the liver and stomach is composed of a stroma containing tubular spaces and large irregular spaces lined or filled with cylindrical cells.

CASE XXXVIII. *Cancer of the stomach, diffuse infiltration.* Male, 57 years old, February 11.

He says he was well until eighteen months ago. Then he began to have dull pain in the epigastrium and occasional vomiting.

One year ago he vomited clotted blood, since then the pain in the epigastrium has been worse and the vomiting more frequent.

He is now emaciated with regurgitation of food, pain and a tumor in the epigastrium.

April 13 he died.

Autopsy. The brain was not examined.

The heart was small and brown. There were old adhesions and fibrous tissue at the apices of the lungs.

The liver was small and contained no tumors.

The spleen and kidneys were normal.

There is a diffuse growth of the whole wall of the stomach from the pylorus to four inches up the œsophagus. All the coats of the stomach are thickened without ulceration or a circumscribed tumor. The growth is composed of large, polygonal nucleated cells, without any definite arrangement.

CASE XXXIX. *Cancer of the stomach in a man twenty-six years old.* Male, 26 years old, November 9.

He says that he was always well until ten weeks ago, then he began to have pain in the epigastrium after eating. The pain continued, grew worse and two weeks ago he began to vomit.

Vomiting of food and coffee-ground matter about once a day continued, on November 9 a tumor could be felt in the epigastrium. He steadily lost flesh and strength and died on Dec. 10.

Autopsy. The brain was not examined.

The heart was small.

There were white, flat nodules in the pulmonary pleura.

The liver and spleen were normal.

The kidneys were small, chronic nephritis.

There were nodules of new growth in the omentum.

The lumbar glands were infiltrated with new growth.

There was a large mass of new growth at the pyloric end of the stomach, infiltrating its wall and projecting inwards in fungous masses. The stomach was dilated and full of food. At one point there is a perforation of the stomach and an erosion of the lower surface of the left lobe of the liver.

The new growth is composed of a stroma containing small, tubular and round spaces filled with small, polygonal nucleated cells.

CASE XL. *Cancer of the stomach and liver, resembling cirrhosis.* Male, 51 years old, Feb. 13.

He says that he was perfectly well until one month ago. Then he began to have pain in the stomach, nausea, swelling of the feet and scanty urine.

Now, he is emaciated, with an enlarged liver, abdominal dropsy, and œdema of the legs, urine 1.028, no albumen.

February 26 he died.

Autopsy. The brain was normal.

There was old thickening of the aortic and mitral valves of the heart.

The lungs were œdematous.

The abdominal cavity was full of serum.

The liver weighed ten pounds and was studded with white nodules.

On the lesser curvature of the stomach, near the pylorus, was a small ulcer with thickened edges. The thickened edges of the ulcer were only hypertrophied mucous membrane. It is only in the floor of the ulcer that there are irregular spaces lined with cylindrical cells.

The nodules in the liver are composed of irregular follicles filled with cylindrical or polygonal cells.

The spleen was small.

The kidneys were small, chronic nephritis.

CASE XLI. *Cancer of the stomach, resembling pernicious anæmia.* Female, 23 years old, Jan. 24, 1889.

In September, 1888, she began to feel weak, with dyspnœa and palpitation on exertion. These symptoms have increased and she has had attacks of syncope. A few days before admission she vomited.

Now, she is somewhat emaciated, very pale, no appetite, pulse 92, breathing 24, temperature 99°. Urine 1.022, trace of albumen. A systolic murmur at the apex of the heart and second left space. She was supposed to have simple anæmia and put on iron and oxygen.

Jan. 28, hæmoglobin 28 per cent., red cells 2,340,000.

Feb. 15, hæmoglobin 18 per cent., red cells 2,224,000.

March 1. She has steadily lost flesh and strength and now vomits.

March 11, hæmoglobin 20 per cent., red cells 1,566,000. She steadily lost flesh and strength with occasional vomiting, and died on March 22.

Autopsy. The brain was not examined.

The lungs were pale and œdematous, with a little red hepatization of the left lower lobe. In both lower lobes were several small, white nodules the size of a pea.

The bronchial glands were not enlarged.

The heart was normal.

The liver was large and contained several large nodules of new growth.

The spleen was normal.

The kidneys were large, smooth and pale.

The uterus was normal.

The stomach was somewhat dilated. At the pyloric end, completely surrounding the stomach and narrowing the pylorus, was a soft, fungous mass of new growth. It follows the type of tubules lined with cylindrical epithelium.

Case I.—Distress after eating and vomiting without disease of the stomach.

Case II.—Stomach symptoms without real disease.

Case III.—Symptoms resembling those of cancer of the stomach; complete recovery.

Case IV.—Gastric symptoms without disease of the stomach.

Case V.—Persistent vomiting without disease of the stomach.

Case VI.—Marked gastric symptoms without disease of the stomach.

Case VII.—Acute, followed by subacute gastritis.

Case VIII.—Acute gastritis, the severe form.

Case IX.—Pyloric gastritis, repeated attacks.

Case X.—Acute gastritis with complications.

Case XI.—Ordinary chronic gastritis; recovery.

Case XII.—Ordinary chronic gastritis; recovery.

Case XIII.—Chronic gastritis with pain and vomiting; recovery.

Case XIV.—Chronic gastritis with severe pain; recovery.

Case XV.—Chronic gastritis, pain, vomiting, emaciation; recovery.

Case XVI.—Chronic gastritis with exudation of serum.

Case XVII.—Headaches with bile in the stomach; recovery.

Case XVIII.—Chronic gastritis with displacement of the stomach and right kidney.

Case XIX.—Chronic gastritis, fatty liver, vomiting of blood.

Case XX.—Chronic gastritis with thickening of the wall of the stomach.

Case XXI.—Temporary dilatation of the stomach.

Case XXII.—Chronic gastritis, dilatation of the stomach; recovery.

Case XXIII.—Chronic gastritis, dilatation of the stomach, tacchycardia; recovery.

Case XXIV.—Chronic gastritis, stenosis of the pylorus.

Case XXV.—Chronic gastritis, stenosis of the pylorus.

Case XXVI.—Tubercular gastritis.

Case XXVII.—Croupous gastritis.

Case XXVIII.—Ulcer of the stomach.

Case XXIX.—Ulcer of the stomach.

Case XXX.—Multiple ulcers of the stomach.

Case XXXI.—Ulcer of the stomach and duodenum, misleading history.

Case XXXII.—Perforating ulcer of the stomach.

Case XXXIII.—Ulcer of the stomach; death from bleeding.

Case XXXIV.—Ulcer of the stomach from an embolus.

Case XXXV.—Cancer of the stomach, large.

Case XXXVI.—Cancer of the stomach, circumscribed tumor.

Case XXXVII.—Cancer of the stomach, general dropsy.

Case XXXVIII.—Cancer of the stomach, diffuse infiltration.

Case XXXIX.—Cancer of the stomach in a man twenty-six years old.

Case XL.—Cancer of the stomach and liver, resembling cirrhosis.

Case XLI.—Cancer of the stomach, resembling pernicious anæmia.

THE LIVER.

Simple, or Catarrhal Jaundice.

Lesions.—There is a catarrhal inflammation of the common bile-duct alone, or in addition there is a catarrhal gastritis, or a catarrhal duodenitis. The inflammation is attended with so much swelling of the mucous membrane of the common bile-duct as to obstruct the flow of bile from the liver into the intestine.

Symptoms.—The patients suffer from general malaise, headache, dullness, sleepiness, or vertigo. The tongue is coated, there is loss of appetite, nausea, and vomiting. There may be a febrile movement. Sometimes there is pain over the duodenum. After one or more days comes the yellow color of the conjunctiva and of the skin, the clay-colored fæces, and the dark-colored urine, with the itching of the skin. The urine, besides the bile-pigment, contains albumin and casts. After the appearance of the jaundice the gastric symptoms may subside.

If the bile-duct alone is inflamed the cases run a mild course. The one symptom is the jaundice. The patients are not confined to bed, the cerebral symptoms are slight, there is no vomiting, and no fever.

If the duodenum is inflamed as well as the common bile-duct the prostration is more marked. There is often a good deal of pain in the epigastrium. Nausea, vomiting, and a febrile movement are absent, or not severe.

If the stomach also is inflamed the condition of the patients is much worse. They are often confined to bed. There may be stupor and delirium. Nausea, vomiting, and a febrile movement are regularly present. The symptoms of the gastritis may precede by several days the appearance of the jaundice.

When gastro-duodenitis with jaundice occurs in a person who already has a cirrhotic or a fatty liver, it is often fatal.

Although these cases are of ordinary occurrence and the conditions seem simple there are features difficult to understand.

The inflammation of the bile-duct, duodenum and stomach are sufficient to account for the pain, nausea and vomiting and the severity of these symptoms is in proportion to the severity and extend of the inflammation.

The retention of bile accounts for the headache and stupor. But the severity of these symptoms is not always in proportion to the evidences of bile retention.

The most obscure feature is the condition of sepsis, with dry tongue, rapid emaciation, delirium and stupor and high temperatures. These patients are for the time very ill, but yet regularly recover.

Treatment.—The most efficient treatment is the administration of alkalies by the stomach and hot water irrigations by the rectum. The feeding is according to the severity of the gastritis and the sepsis.

CASE I. *Catarrhal jaundice with high temperatures.* Male, 27 years old, November 30.

Nov. 26 he was seized with repeated chills, pains all over the body, nausea and marked prostration.

Nov. 30, temperature 105°, pulse 104, tongue dry, skin a little yellow, no vomiting, bowels loose, urine contains bile-pigment, no albumen, liver of normal size.

Dec. 1, temperature 100° to 102°.

Dec. 3, temperature 99° to 100°, the jaundice is increasing.

Dec. 6, temperature 99°, the jaundice is now very marked.

Dec. 17, the jaundice is fading and the patient sitting up.

He was not entirely well until January 4.

CASE II. *Gastro-duodenal jaundice with marked diminution in the size of the liver.* Female, 24 years old, June 28.

She was well and strong until three weeks ago. Then she was suddenly attacked with pain in the epigastrium, followed by vomiting. The pain and vomiting continued and on June 27 she became jaundiced.

June 28, she was feeble, but not emaciated, deeply jaundiced, vomited frequently a thin, green fluid, the epigastrium was tender, the bowels constipated.

She continued to vomit and to look badly, temperature 99° to 100°, pulse 120 to 130.

July 1. She was looking badly, I could not make out any liver dulness in the mammary line. She was put on the steady use of bicarbonate of soda with milk.

July 3, the vomiting was better.

July 7, very little vomiting, there was some dulness over the liver, I could not map it out, although the abdomen was thin.

July 10, no more vomiting, temperature normal, jaundice fading, better in every way.

July 13. The upper line of liver-dulness in a line between the sternum and nipple begins 3½ inches above the free border of the ribs, the lower border is one inch above the free border of the ribs.

July 18, eating solid food, out of bed, no jaundice.

Aug. 12, the liver-dulness in a line between the sternum and nipple extended from the top of the fourth rib to the free border of the ribs.

In patients in whom jaundice persists for months the cause of the jaundice is difficult to make out. There are a few cases in which the cause is a chronic gastro-duodenitis.

Case III. *Chronic gastro-duodenitis with jaundice; improvement.* Female, 50 years old, Nov. 25, 1902.

Six months ago she began to have nausea, vomiting once a week, and two months later jaundice. The yellow color of the skin, dark urine and white fæces have continued ever since; there has been no pain, she has lost 35 pounds in weight.

Nov. 25, the liver is somewhat enlarged downward, washing of the stomach gives undigested food and mucus.

Dec. 2, the nausea and jaundice continue unimproved.

Dec. 9, she is no better, to wash the stomach every day.

Dec, 16, the nausea is better, the jaundice continues.

Dec. 18. There is now a little bile in the fæces.

Dec. 23. She continues to improve, the color of the skin is lighter, there is more bile in the fæces.

Dec. 30. She continues to improve, there is no bile-pigment in the urine.

Jan. 20, 1903. She seems to be well, except that there is still a tinge of yellow in the skin.

May 8. She has continued to feel well, the liver has returned to its natural size, the skin is of a light yellow color, the urine contains a little bile-pigment, the fæces are of normal color.

After this the woman did not come back.

Case IV. *Fatal jaundice caused by chronic duodenitis.* Male, 51 years old, June 10.

Two months ago he began to have a feeling of oppression in the right hypochondriac and umbilical regions. Three weeks later jaundice began and increased.

June 10, he is fairly nourished, no appetite, bowels regular, fæces clay-colored, pulse and temperature normal, urine bile stained, liver enlarged, general deep yellow jaundice of the skin.

His color gradually became darker until he was nearly black, he steadily lost flesh and strength, had frequent epistaxis and died December 23.

Autopsy. The pia mater was thickened and adherent to the dura mater with an increase of serum.

The heart was normal, the aorta was athromatous.

The pleura of the left lung was coated with recent fibrin, in the upper lobe there were small fibrous and cheesy nodules.

The spleen and kidneys were normal.

The supra-renal capsules were normal.

The stomach was small and empty. Its entire mucous membrane was blackish and thickly coated with mucus and pus. There was an extensive growth of fibro-cellular tissue between the gastric tubules with atrophy of the tubules.

The mucous membrane of the duodenum was thickened, black and coated with mucus.

The liver was of ordinary size and consistence, bile stained. The gall-bladder was dilated and distended with normal bile. The hepatic and common ducts were dilated, but not the ducts within the liver. Pressure on the gall-bladder or common duct did not cause the bile to flow into the duodenum, but a large director could be passed through the hepatic and common ducts.

The entire length of the pancreatic duct was dilated, but a director could be passed through it.

There were several large, cheesy glands surrounding the bile-ducts.

ACUTE DEGENERATION OF THE LIVER.

Synonyms.—Acute yellow atrophy of the liver. Malignant jaundice.

As the result of some unknown poison there is a very rapid and fatal degeneration of the hepatic cells and the renal epithelium, with general sepsis.

Lesions.—The liver may at first be somewhat enlarged and then become smaller, or it becomes smaller from the first. The diminution in size is very rapid and goes on until the liver only weighs one or two pounds. These very small livers are yellow and flabby.

In other cases the liver is not much diminished in size, its consistence is firm, its color is mottled red and yellow.

In all cases there are profound changes in the hepatic cells. They become swollen, coarsely granular, and finally disintegrate and break down.

The spleen is large and soft. The stomach is the seat of acute catarrhal inflammation.

In the intestines the solitary and agminated glands are swollen.

The kidneys are the seat of very intense acute degeneration. In most of the tubes the epithelial cells are dead and detached from the walls of the tubes.

The muscles of the heart undergo fatty degeneration.

There are extravasations of blood in the skin, the mucous membranes, and the serous membranes.

Causes.—The disease has been observed in persons between the ages of one and sixty-nine years, but it is especially common in persons between twenty and thirty years of age. It is more common in women than in men. It has been seen a number of times in pregnant women.

Symptoms.—The invasion of the disease is gradual, or sudden.

If it is gradual, the patients suffer from nausea, occasional vomiting, general malaise, or sometimes jaundice, for from three days to three weeks.

If it is sudden, the patient at once becomes seriously ill with a rapid development of all the symptoms of the disease.

When the disease is established, there is frequent vomiting, first of food then of brownish fluid, coffee-ground matter and blood. There is a diarrhœa with clay-colored fæces, and later bleeding from the intestines. Jaundice in two-thirds of the cases appears early in the disease, in one-fourth of the cases from the fifth to the seventh day, in a few cases not until one or two days before death. In very rapid cases there may be no jaundice. There may be pain over the region of the liver. There is a febrile movement during the active period of the disease, but the temperature runs an irregular course, sometimes very high, sometimes falling below the normal. The pulse becomes rapid and feeble.

There are cerebral symptoms—headache, alternating stupor and delirium, general convulsions, and coma.

The tongue becomes brown and dry, the prostration is very marked.

The liver may at first be enlarged and then become smaller, or it may become smaller from the outset. The diminution in size, as we mark it out by percussion, is very rapid, and finally the liver dulness may entirely disappear. In some cases, however, the liver remains throughout the disease of nearly normal size.

The urine contains bile-pigment, albumin, casts, blood, leucin, and tyrosin. The urea and uric acid are diminished in quantity.

There are often hemorrhages from the stomach, intestines, and kidneys, and in the skin.

The ordinary duration of the active symptoms is a week, but a prolonged period of invasion may lengthen the whole time of the disease to four weeks, and the bad cases may die within twelve hours.

The prognosis of the disease is very bad.

CASE V. *Acute yellow atrophy of the liver.* Male, 22 years old, physician serving as interne in a New York hospital.

Nov. 25, after having passed a restless night, he complained of nausea and pain in the bowels. In the evening he felt better and slept part of the night.

Nov. 26 he complained of headache and pain in the back, at 9 A. M. he had a chill followed by a temperature of 104°, pulse 144, followed by sweating. In the evening the temperature was normal.

Nov. 27, at 8.45 A. M., a severe chill followed by a temperature of 106°, the urine was scanty and high colored, but contained no albumen, he vomited several times, in the evening the temperature was normal.

Nov. 28, vomiting and retching continued, the eyes were a little yellow, the face was haggard, tongue dry, pulse 130, temperature 98°.

Nov. 29, vomiting continued, pulse 70, temperature 98°, general jaundice, pain and tenderness over the liver, delirious. In the afternoon he vomited coffee-ground matter, the urine contained albumen and blood, temperature remained at 98°.

Nov. 30, he had a better night, the vomiting stopped, delirium continued, temperature 99°, pulse 120; he took milk and retained it. The liver-dulness was diminished. He became steadily weaker during the day and died at 6 P. M.

Autopsy. The body was deeply jaundiced. There were ecchymoses in the visceral pericardium, otherwise the heart was normal.

The lower lobes of both lungs were congested. In the left lower lobe there was a yellow infarction as large as an English walnut.

The liver weighed two pounds ten ounces, it was of firm consistence, of light yellow color, mottled by red spots at the centres of the acini. The gall-bladder contained a small quantity of thick, black bile. The liver cells are swollen and filled with small granules, some of them are broken down into granular matter.

The stomach contained a little blackish fluid, its mucous membrane was pale.

The duodenum was pale.

The spleen was of medium size, not softened.

The kidneys were large, the renal epithelium throughout was swollen and granular. The large tubes in the pyramids were packed with blood and granular matter.

CASE VI. *Acute yellow atrophy of the liver.* Male, 19 years old, drug clerk, December 23.

Two weeks ago he was taken sick with loss of appetite, pain in the epigastrium and general malaise. After a week he became jaundiced, feeble, urine dark colored, pulse slow, temperature normal, fæces clay colored, on December 23 liver percussed six and one-half inches in mammary line.

Dec. 28, delirious, vomiting, pulse 80, temperature 99°.

Dec. 30, pulse 130, temperature 102° to 105°. He died comatose.

Autopsy. The body was deeply jaundiced.

The brain was normal.

The heart was normal.

The lungs were congested.

The spleen was large and soft.

The liver weighed two and one-half pounds, flabby, of normal color.

The liver cells were distended with small shining granules and oil globules so that they looked black by transmitted light; some of the cells were broken down. In many sections were to be seen a large number of tubular follicles looking like hypertrophied bile-ducts.

In the pericardium and peritoneum were large ecchymoses.

The stomach and duodenum contained black fluid.

The kidneys were swollen and yellow.

Case VII. *Acute yellow atrophy of the liver.* Female, 25 years old, prostitute, intemperate.

She had been drinking hard for two weeks.

About one week before death she began to have vomiting, jaundice and prostration.

Autopsy. The body was deeply jaundiced.

The aortic valves of the heart were thickened and insufficient.

The lungs were small and congested.

The liver weighed three pounds, was of yellow—mottled with red—color, the gall-bladder was half full of black bile. The liver cells were distended with coarse granules.

The spleen was normal.

The stomach contained thick, black fluid.

The kidneys were of medium size, the renal epithelium was granular and broken down.

There were large ecchymoses in the pericardium and peritoneum.

Case VIII. *Acute yellow atrophy of the liver.* Male, 35 years old, sailor; has had yellow fever some years ago.

Eleven days before death he began to feel sick and have irregular pain. Six days before death he had a chill. Four days before death he looked very ill with general pains, vomiting, rapid pulse and jaundice.

He became steadily weaker and died comatose.

Autopsy. The body was deeply jaundiced.

The brain was normal.

The heart was soft and flabby.

The lower lobes of the lungs were congested.

The liver weighed four pounds, was of greenish yellow color, soft and friable. The liver cells were completely broken down into granular matter. There was no pigment.

The spleen weighed two pounds, was soft, contained no pigment.

The kidneys were large, the epithelium was swollen and granular.

CASE IX. *Acute yellow atrophy of the liver.* Female, 35 years old, January 13.

Jan. 10 she became nauseated and jaundiced.

Jan. 12 she was delivered of a seven months' child. She did well for twenty-four hours after the birth of the child, then she began to vomit and the next night became delirious.

Jan. 13, she was semi-comatose, jaundiced, urine bile stained, a little albumen, no casts.

Jan. 14, temperature 103° to 104°, pulse 104 to 136, the liver measured 3½ inches in the mammary line.

Jan. 15, temperature 105° to 107°, pulse 156 to 176, liver measured 2½ inches in the mammary line, at 4 P. M. she died.

Autopsy. All the tissues were yellow.

The brain was not examined.

The heart was normal.

The lower lobe of the right lung was hepatized.

The stomach and intestines were normal.

The spleen was of medium size. The liver weighed two pounds. It was of bright yellow color, smooth, soft and flabby. The acini were distinct, their centres opaque yellow, the peripheries more translucent and gray. At the centres of the acini the liver cells preserve their shape although they are granular and fatty. At the peripheries of the acini the liver cells are lost in a mass of granular matter and free fat. In some places there are collections of small, round cells at the peripheries of the acini.

The kidneys were of medium size.

The malpighian bodies were unchanged. In the cortex the epithelium of the straight and convoluted tubes is granular, swollen and in places detached, oil globules are mixed with the epithelium.

In the middle and upper part of the pyramids the straight tubes are filled with detached and granular epithelium. Near the apices most of the tubes contain hyaline casts.

CASE X. *Acute yellow atrophy of the liver.* Female, 29 years old, September 29.

Three weeks ago she began to have headache, vomiting, prostration, and a few days later jaundice.

Sept. 29, headache, pain in the epigastrium, vomiting, marked jaundice, pulse 68, temperature 98°.

Oct. 3 to 5 she was better, with less vomiting.

Oct. 6, temperature 100°, more vomiting, increasing jaundice, delirium and stupor, liver diminished in size, urine contains albumen and leucine.

Oct. 9, ecchymoses in skin, temperature 99°, coma, death.

Autopsy. The body was deeply jaundiced. The muscular fibres of the heart were fatty.

The lungs were congested.

The liver weighed two pounds, was soft, mottled red and yellow, the liver cells were distended with coarse granules.

The stomach and intestines were normal.

The kidneys were large, yellow, the epithelial cells throughout swollen, granular and detached.

CIRRHOSIS OF THE LIVER.

Synonym.—Chronic interstitial hepatitis.

Lesions.—There is a chronic productive inflammation of the stroma of the liver with more or less degeneration of the hepatic cells. The new tissue follows the distribution of the stroma of the liver. It surrounds large or small groups of acini, or grows diffusely between the liver-cells, or is in the form of large bands and masses. According to the distribution of the new connective tissue, the liver is coarsely or finely nodular, or smooth, or lobulated. The liver-cells may become atrophied or fatty. The small veins and bile-ducts are often obliterated. The larger bile-ducts may be the seat of catarrhal inflammation. The liver remains of normal size, or becomes small or large. We find after death: small nodular livers, small smooth livers, large nodular livers, large smooth livers.

The obstruction to the passage of the blood from the portal vein through the liver causes the spleen to become large and hard, the mucous membrane of the stomach and small intestines to be congested and coated with mucus, the peritoneal cavity to contain serum.

Causes.—The disease belongs to adult life, and is usually caused by alcohol or syphilis. It is, however, occasionally seen in children, and may occur without discoverable cause.

Ideas concerning the nature of cirrhosis have become unnecessarily confused. It has been taught that the primary change is a degeneration of the liver cells and that this is followed by the growth of connective tissue. The attempt has also been made to separate the large from the small cirrhotic livers as a separate variety. As a matter of fact cirrhosis of the liver behaves like the other chronic productive inflammations and is often associated with them. The growth of new connective tissue is constant, the degeneration of the liver cells is variable. The disease is essentially the same whether the livers are large or small, smooth or nodular.

Symptoms.—These depend on:

1. The disturbance of the bile-producing function of the liver, with the consequent digestive disturbances and loss of nutrition.
2. The accompanying chronic gastritis.
3. The interference with the passage of the blood through the liver and the consequent dropsy.
4. The development of jaundice.
5. The existence of a complicating nephritis, arteritis, or emphysema.

Physical Signs.—These depend on the changes in the size of the liver and the smooth or nodular character of its surface; on the enlargement of the spleen; and on the presence of fluid in the abdominal cavity.

The course of the disease varies in different persons.

1. There are persons who have cirrhosis of the liver for a number of years without any symptoms to call attention to that organ.

2. In a good many cases of cirrhosis the principal symptoms are those of the accompanying chronic gastritis. This gastritis seems to be partly due to chronic alcoholism, or some other ordinary cause, partly to the chronic congestion, caused by the cirrhosis. It behaves like any chronic gastritis, but is often severe and rebellious to treatment.

The patient may also vomit blood in large or small quantities. This bleeding may come from dilated veins in the lower end of the œsophagus.

3. The anatomical changes in the liver after a time affect the functions of the organ in such a way that the patients steadily lose flesh and strength. This loss of nutrition is a very constant symptom, and seems to have a large share in causing the death of the patients. It is most frequently associated with dropsy, but may exist and cause death without any dropsy at all.

4. Ascites is one of the ordinary features of cirrhosis; general dropsy occurs less frequently. When the dropsy has once appeared it is apt to continue and the patients get steadily worse. But occasionally even a large abdominal dropsy can be gotten rid of and the patient do well for a number of years.

5. Patients with cirrhosis of the liver are liable to slight attacks of catarrhal jaundice, and to severe attacks of gastro-duodenitis with jaundice. These latter attacks are attended with fever and cerebral symptoms and are often fatal.

6. With the large, nodular, cirrhotic livers (hypertrophic cirrhosis) there often occurs a very fatal form of jaundice. It behaves like a bad obstructive jaundice, although the hepatic and common bile-ducts are pervious.

The patients have pain over the liver, vomiting, constipation, a constantly increasing jaundice, a febrile movement, gradual emaciation, and cerebral symptoms. All of them die.

The prognosis of cirrhosis is unfavorable, but yet in many cases the course of the disease is slow, and in some it is arrested altogether. The patients have no symptoms, although the cirrhosis still exists.

Treatment.—In many cases of cirrhosis we only treat the chronic gastritis. This is done in the usual way by regulation of the diet and mode of life and by lavage. But it is found that if the cirrhosis progresses it becomes more and more difficult to control the gastritis.

In the cases with marked loss of nutrition the use of alcohol and of tobacco is to be prohibited. The patients should lead, as far as possible, an out-door life. It may be necessary to relieve constipation and to give drugs which increase the production of bile.

If there is a distinct history of syphilis it may be necessary to give mercury and the iodide of potash.

Moderate quantities of fluid in the peritoneal cavity can be gotten rid of by diuresis and by purging, but when the abdomen is distended with fluid it is necessary to tap.

CASE XI. *Cirrhosis, small and smooth liver, loss of nutrition and dropsy.* Male, 56 years old, an habitual drunkard.

He has had frequent attacks of headache and nausea, but has considered himself to be in fair health until about four months ago.

At that time he was said to have had typhoid fever. Four weeks after his convalescence his feet and abdomen began to swell. He became much emaciated and had frequent attacks of epistaxis.

He is now feeble, emaciated, jaundiced with dropsy of the legs and abdomen. He remained in this condition for six weeks and then died.

Autopsy. The liver weighed two pounds. Its surface and cut surface were smooth. There was a large growith of connective tissue around and between the acini, but only to a small extent between the liver cells. Only a few of the liver cells were fatty, the rest were of normal size and appearance.

CASE XII. *Cirrhosis of the liver, large and smooth liver, loss of nutrition, dropsy and jaundice.* Male, 52 years old, October 20.

For fifteen years he has been a steady gin drinker.

For ten years he has had moderate dropsy of the feet and legs.

Two years ago he began to vomit and to lose flesh and strength.

Six weeks ago he developed jaundice and ascites.

One week ago he was run over by a horse and since then he has been in bed.

Oct. 20, he was unconscious, with general dropsy, labored breathing, urine 1.012, no albumen, normal quantity. He continued drowsy until he died on Oct. 27.

Autopsy. The brain showed a marked increase of serum.

The heart was normal.

The lungs were congested and œdematous with serum in both pleural cavities.

The liver weighed five pounds, was smooth, hard, yellow, with a diffuse growth of connective tissue.

The spleen was normal.

The kidneys weighed 12 ounces, the capsules were adherent, lesions of chronic nephritis, not advanced.

CASE XIII. *Cirrhosis, hæmatemesis, no ascites.* Female, 31 years old, November 10.

Temperate, has had good health.

Three years ago she first noticed some enlargement of the spleen.

Two years ago she began to have a troublesome cough.

Eight months ago she vomited blood and after that was feeble, coughed incessantly and was miserable for four months. Then she was better, went to work and continued to be pretty well until two weeks ago. Then the cough became troublesome and the spleen painful.

Nov. 10, cough, no physical signs over the heart or lungs, the spleen was large, tender and painful, temperature 102°, urine, no albumen or casts.

Nov. 12, temperature 101° to 102°, tongue brown and dry, nausea.

Nov. 13, stupor alternating with delirium, temperature 99° to 103°. She continued in the condition until Nov. 23. Then she was better, temperature normal, appetite returning, she was out of bed.

Dec. 18, vomited about an ounce of bright blood. After this she became feeble again.

Dec. 30. She vomited a pint of blood in the morning and again in the evening.

January 10, she vomited blood and could not retain food.

Jan. 17, she vomited a pint of blood.

Jan. 20 and 21, she continued to vomit blood, became feeble and drowsy.

February 4, she died.

Autopsy. The brain was not examined.

The heart was normal.

In the lungs there were scattered, old patches of black fibrous tissue.

The liver weighed three pounds and was nodular. There was a growth of connective tissue around groups of acini with fatty infiltration of the liver cells.

The spleen weighed two pounds, simple hypertrophy.

The mucous membrane of the stomach was pale and coated with mucus.

CASE XIV. *Syphilitic cirrhosis, chronic peritonitis, no tapping.* Male, 32 years old, October 16.

Three years ago he had a chancre.

One year ago he had sore throat.

For fifteen months he had ulcers on the legs.

Four weeks ago he developed cough and dyspnœa.

One week ago he began to have ascites.

Oct. 16, he is feeble and emaciated, temperature 98°, hydrothorax, ascites, œdema of the legs, large nodular liver, pain over the abdomen, albumen in the urine.

October 30, marked abdominal pain and tenderness, delirious, temperature 103°, pulse 104, breathing 50.

Oct. 31 he died.

Autopsy. The brain was not examined.

The aortic and mitral valves were a little thickened.

The pleural cavities were nearly full of serum, the lungs compressed and unaerated.

The liver was large and nodular, no changes in the gall-bladder or bile-ducts.

The spleen was large.

The stomach and kidneys looked normal.

The peritoneal cavity was full of reddish serum. The whole peritoneum was thickened by a diffuse growth of loose vascular connective tissue. This was coated with fibrin.

CASE XV. *Cirrhosis of the liver, fatal jaundice.* Male, 51 years old. May 13.

He has been gouty for several years, confined to his room much of the time and always intemperate.

Four weeks ago he began to have jaundice with some œdema of the legs.

May 13, pulse 80, temperature 99°, general jaundice, urine bile stained.

The jaundice steadily increased, he became first stupid, then unconscious and died on May 18.

Autopsy. The brain was not examined.

Around the heart was an increase of sub-pericardial fat, the aortic and mitral valves were a little thickened, both ventricles were moderately hypertrophied.

The lungs were small, the bronchi congested and coated with mucus.

The liver weighed eight pounds, was yellow, nodular, cirrhotic, no obstruction of the large bile-ducts.

The spleen weighed one pound.

The mucous membrane of the stomach was coated with mucus.

The kidneys showed the lesions of chronic nephritis.

CASE XVI. *Cirrhosis of the liver, fatal jaundice.* Male, 65 years old, October 31.

He gives no history of intemperance or syphilis.

One year ago he began to have a diarrhœa which has continued ever since.

For three weeks he has had nausea and vomiting and for a week he has seemed to be very stupid.

Two weeks ago he became jaundiced and his abdomen began to swell. He is now deeply jaundiced, emaciated, the liver enlarged, serum in the abdominal cavity, mind dull and wandering, pulse 120, temperature 99°, vomiting, fæces white, urine bile stained, no albumen. He became weaker and died November 8.

Autopsy. The body was emaciated and deeply jaundiced.

The brain was not examined.

The heart was normal.

There was serum in both pleural cavities, the lungs were congested and œdematous.

The peritoneal cavity contained serum.

The spleen was large .

The liver was of medium size, it extended a hand's breadth below the free surface of the ribs, its surface was finely nodular, it was bile stained and very hard. There was a diffuse growth of connective tissue surrounding groups of acini, single acini and extending between the liver cells. The liver cells were swollen and granular. The large bile-ducts in the liver, the hepatic duct and the common duct were of natural size and appearance. The cystic duct was obliterated, the gall-bladder was small, its wall was thickened, it contained inspissated bile.

The mucous membrane of the stomach was blackish.

The wall of the small intestine was thick and stiff. This seemed to be due to a thickening of the muscular and connective-tissue coats, there was no change in the glandular coat.

The wall of the colon was thickened, there was an infiltration of pus cells in the connective-tissue coat.

The kidneys looked normal.

CASE XVII. *Thickening of the capsule of the liver with dropsy and vomiting of blood.* Female, 30 years old, September 7; intemperate.

Two months ago she began to have chills and fever every other day.

One month ago the chills stopped and the feet and abdomen became swollen.

Sept. 28 the abdomen was tapped.

Oct. 4, since the tapping there has been a little fever and occasional vomiting.

Oct. 8 she suddenly vomited two ounces of clotted blood. The vomiting of blood was repeated several times after this.

Oct. 17 she died.

Autopsy. The brain was not examined.

The heart was normal.

The lungs were covered with old adhesions.

The capsule of the liver was very much thickened and adherent to the surrounding viscera. The liver was compact and smooth without any new growth of connective tissue. There were calculi in the gall-bladder.

The stomach contained black blood, there were no ulcers.

The kidneys were small and hard.

AMŒBIC ABSCESS OF THE LIVER.

Amœbic abscesses of the liver are of not infrequent occurrence in New York. The infection may come first in the colon and later in the liver, or there may be a primary infection of the liver with no changes in the colon.

Lesions.—The amœbic abscesses usually follow one of four anatomical forms.

1. They resemble ordinary abscesses in any part of the body. There are one or more abscess cavities of considerable size filled with well-formed pus, and their walls infiltrated with pus-cells. The abscesses may be situated in any part of the liver. This is the least fatal form of liver abscess.

2. There are one or more cavities in the liver with ragged, irregular, necrotic walls, and containing a thin reddish, or brownish, or yellow fluid, with fragments of dead liver-tissue, but very few pus-cells.

3. A considerable area of liver-tissue is dead, with little cavities filled with pus or puriform fluid scattered through it.

4. The entire liver is studded with small abscesses, some of them too small to be seen with the naked eye.

In all these abscesses the amœba coli may be found. In the smallest abscesses the first change is a necrosis of the liver-tissue.

Whether the different anatomical characters of the abscesses are due to the addition of bacterial infection to the amœbic infection has not been worked out.

If the abscesses are situated near the surface of the liver, there will be exudative inflammation and the formation of adhesions.

If left to themselves the abscesses usually increase in size until they perforate through the diaphragm into the lung, the pleura, or the pericardium; or into the stomach, the intestines, the peritoneal cavity, or the pelvis of the right kidney; or through the anterior abdominal wall.

Even without perforation there may be inflammation of the right pleura, or abscesses in the right lung.

Symptoms.—In some of the patients there is the history of one or more attacks of colitis, then the invasion of the liver abscess with chills, fever and pain.

In some patients without any previous symptoms there is a sudden invasion with chills, fever and pain over the liver.

In some patients the invasion is gradual and obscure. The patients feel chilly and feverish, lose their appetite, feel sick, have nausea and vomiting, but do not go to bed for several days; after they go to bed the fever continues with chills and sweating. The nausea and vomiting continue, they lose flesh and strength, there are pain and tenderness over the liver.

The physical signs vary with the position and size of the abscess. Large abscesses in the lower part of the liver cause considerable enlargement downward into the peritoneal cavity.

Abscesses in the upper part of the right lobe of the liver give dulness over the lower part of the right pleural cavity. Some abscesses in the right lobe only give a fulness of the right hypochondrium without enlargement of the liver either upward or downward. The most obscure cases are those of small abscesses scattered through the liver.

Treatment.—The success of treatment depends on the position and character of the abscess. If of moderate size and a single cavity containing pus it can be cured by aspiration. Larger abscesses need to be opened and, if they project into the pleura, this may be difficult. Multiple abscesses and diffuse necrosis are not amenable to treatment.

CASE XVIII. *Abscess of the liver, one aspiration; recovery.* Male, 32 years old, October 21, 1887.

He was in his usual health and working as a laborer until Oct. 7. Then for a week he had four or five watery stools every day, but continued to work.

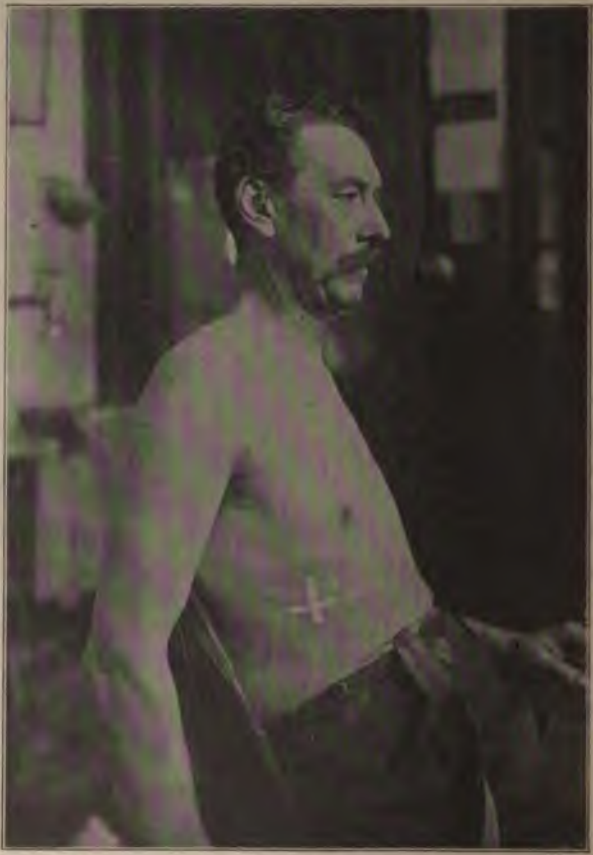
Oct. 9, about 9 A. M., he had a chill for ten minutes and continued all day to feel chilly and miserable with vomiting and pain over the liver.

Oct. 10 he tried to work, but had to stop on account of the pain. The pain is referred to the liver and is very severe.

Oct. 21, pulse 88, breathing 24, temperature 100°, bowels constipated, urine no albumen, no physical signs over the heart or lungs, marked pain and tenderness over the liver. The liver is not enlarged upward or downward, but the hypochondriac region is too full.

Oct. 24 I aspirated through the seventh intercostal space a little in front of the anterior axillary line and drew off 100 cc. of pus.

FIGURE 1.



CASE XIX. AMOEBIC ABSCESS OF THE LIVER.

He had no further symptoms, ate beefsteak on Oct. 26, was out of bed on Nov. 8 and discharged on Nov. 18.

CASE XIX. Figure 1. *Abscess of the liver, one aspiration; recovery.* Male, 46 years old; perfectly well until November 2.

Then he had an attack of severe pain extending from the right shoulder downward to the abdomen with a marked chill. For the three following nights he had chills followed by sweating. The pain has persisted, he cannot rest on the right side, the bowels have been regular.

Nov. 13, he is pale, tongue coated, moderate prostration, temperature 99°, urine 1.020, no albumen, no physical signs over the lungs or heart. The liver is not enlarged upward or downward, but the resonance over it is flatter than it should be. There is no bulging of the intercostal spaces, no pain or tenderness, but a loud friction sound can be heard over the liver.

Nov. 13 to 22, a septic temperature of 99° to 103°.

Nov. 22 I aspirated at the point marked in Figure 1 and got 200 cc. of reddish pus which contained pus cells, liver cells and amœbæ.

Dec. 2 he was out of bed.

Dec. 14 he was discharged well.

CASE XX. *Amœbic abscess of the liver, allowed to run its course; death.* Female, 42 years old, August 9.

One year ago she had severe pain in the right side below the free border of the ribs, lasting for three months, and then gradually disappearing. She was well after this until last May, when she was suddenly seized with severe pain over the liver, lasting for two hours. This pain has continued ever since.

In July a small tumor appeared in the place where she felt the pain. This tumor has gradually increased in size. She has been having an afternoon fever with sweating.

Aug. 9. It is evident that the swelling is an abscess of the liver. The bowels are constipated.

Aug. 12. Twelve ounces of pus were aspirated from the abscess.

Aug. 20, the abscess was again aspirated.

Aug. 26, the abscess perforated and discharged through the skin.

Sept. 5, she began to have a diarrhœa, which continued until she died. The discharge from the abscess gradually diminished. She ran a septic temperature between 96° and 102°.

Oct. 14 she died.

Autopsy. The brain was not examined.

The heart was normal.

The bronchi contained muco-pus.

In the liver there were two large abscesses. The one which had discharged was empty and apparently healing. The other was of large size.

The kidneys and spleen were normal.

The colon from six inches above the anus to the cæcum was studded with small round ulcers with overhanging edges.

At the cæcum, just above the appendix, a larger ulcer has nearly perforated and caused a general peritonitis.

CASE XXI. *Amœbic abscess of the liver, incision; recovery.* Male, 42 years old, June 5.

Ten months ago he had an attack of dysentery, 6 to 7 passages daily of mucus and blood. Since then he has had several passages a day of loose fecal matter and mucus, but no more blood.

For four months he has vomited greenish matter every morning.

June 5, he is fairly nourished, pulse 104, temperature 99°, tongue coated and moist, the liver extends two inches below the free border of the ribs, there are ulcers on the posterior wall of the rectum.

Aug. 1, the liver extends three inches below the free border of the ribs, is tender and painful. The diarrhœa has stopped.

Aug. 7, the liver now extends down to one inch above Poupart's ligament.

Aug. 17, I made an incision in the right lumbar region into the abscess and put in a drainage tube. Eighty-six ounces of thin, dirty pus escaped.

Aug. 31, the abscess is washed out daily with a solution of chloride of zinc.

Oct. 11, he goes out of doors.

Dec. 26, the abscess is closed, the bowels are regular, he is well.

CASE XXII. *Abscess of the liver, rupture through the diaphragm; death.* Male, 19 years old, June 7, 1881.

May 31 he had an attack of severe pain about the umbilicus, lasting for several hours. Since then he has had repeated attacks of the same pain, bowels constipated.

June 7, a well-nourished boy, does not look seriously ill.

there are no physical signs over the heart or lungs, the liver extends from the top of the fifth rib to one inch below the free border; he has paroxysms of pain about the umbilicus.

June 9, the bowels moved freely with castor oil.

June 11, two chills, looks badly, tongue brown and moist, face flushed, vomiting, constant pain about the umbilicus, temperature 100° with morning remissions. I thought he had typhoid fever.

June 23, he has continued in the same typhoid state, with frequent attacks of pain.

Then his temperature fell to 98°, the pain diminished, he gained a little strength and left the hospital on August 22.

September 1 he came back. He has no more pain, no fever, is very much emaciated but not in bed all the time.

He has a chronic fecal diarrhœa, no physical signs. I could not make a diagnosis.

Sept. 30 he left the hospital.

Oct. 8 he returned. No pain, no vomiting, temperature 100° diarrhœa, no albumen in urine.

November 18 he began to spit up large quantities of foul pus and continued to do so until he died on December 12.

Autopsy. The body was much emaciated.

The brain was not examined.

The heart was normal.

The lower third of the right pleural cavity was shut in by adhesions and full of pus. I did not find a perforation of the lung. The bronchi were coated with muco-pus.

The liver extended one inch below the free border of the ribs. In the right lobe there were several large abscesses, one of them had perforated through the diaphragm. All about the abscesses there was a growth of new connective tissue studded with small foci of pus.

The spleen was normal.

The kidneys looked normal.

The stomach was coated with mucus.

The small intestine was normal.

The lower portion of the colon was thickened and coated with mucus.

Case XXIII. *Abscess of the liver, secondary pericarditis, no intestinal lesion.* Male, 20 years old, October 30.

Twelve days ago he was suddenly seized with pain and tenderness in the epigastric and right hypochondriac regions.

This was followed by fever, headache, loss of appetite, constipation, no chill, no jaundice, no diarrhœa or dysentery.

Oct. 30, he is emaciated, bowels constipated, temperature 104°, pulse 84, urine contains albumen, liver large and tender.

Nov. 7, the fever and enlargement of the liver continue, there is a little jaundice.

Nov. 14, the abscess is pointing one inch below the ensiform cartilage.

Nov. 18, the abscess was opened and ten ounces of good pus discharged.

Nov. 24, the abscess is discharging, the temperature is normal.

Nov. 30, the temperature is rising.

Dec. 7, fever, profuse night sweats.

Jan. 18, after vomiting, he was suddenly seized with severe pain in the left side, precordial oppression and great dyspnœa. There was diffuse pulsation and a friction sound over the precordium.

Jan. 19 he died.

Autopsy. The brain was normal.

The pericardial sac was distended with turbid serum, and coated with fibrin and pus. The heart was small, its walls thin, its valves normal.

The lungs were œdematous.

There were two abscesses in the upper part of the left lobe of the liver, which have perforated the liver so as to form a purulent cavity shut in by adhesions of the lung, pericardium, diaphragm and anterior abdominal wall.

The stomach, intestines, kidneys and spleen were normal.

CASE XXIV. *Small abscesses of the liver, perforation into the pleural and peritoneal cavities.* Female, 43 years old, September 28.

Two weeks ago chilliness, followed by fever, headache, loss of appetite and constipation.

Nine days ago a distinct chill; more fever and pain over the liver.

Sept. 28, she does not look very sick, temperature 100°, pulse 97, pain over the liver, an eruption on the abdomen like that of typhoid.

Oct. 5, the pain over the liver has continued, temperature 100° to 102°, tongue dry and brown, a fluctuating tumor, like an abscess, can be felt in the right side between the ribs and the ilium.

Oct. 8, the abscess was aspirated and two ounces of thin pus removed.

Oct. 14, she was suddenly much worse, with pain in the right side, collapse and death in a few hours.

I had supposed all along that she had typhoid fever with complicating abscesses.

Autopsy. The liver was of medium size, in the right lobe were three small abscesses. One of these communicated with an abscess shut in by adhesions in the peritoneal cavity between the liver and the ileum. The other two abscesses had perforated the diaphragm into the right pleura.

The right pleura contained purulent serum compressing the lung.

The heart, stomach, spleen and intestines showed no lesions.

CASE XXV. *Multiple small abscesses of the liver.* Female, 30 years old, living at Orange, New Jersey.

In November, 1902, she had an attack of colitis with tenesmus and passages of mucus, which lasted two weeks.

In December, 1902, she had a similar attack lasting for two weeks.

In January, 1903, she had a similar attack which is still going on.

January 23 I first saw her. The woman was well nourished, and did not look very sick; temperature 99° in the morning and 101° in the afternoon.

There was a friction sound over the lower part of the right lung behind.

The liver was not enlarged, but there was tenderness over it in front. There are about seven passages a day, composed of a little fecal matter, mucus and small clots of blood. I made the diagnosis of amœbic colitis with a small abscess in the upper part of the right lobe of the liver.

Jan. 26. She looks and feels better; temperature the same, stools the same, liver not enlarged, no pain or tenderness.

Jan. 30. Her condition is the same, she has a clean tongue and good appetite.

Feb. 8, she does not look as well, but the symptoms are the same.

Feb. 14. During this week she has been rapidly getting worse, temperature 99° to 102°, she looks septic. The liver has become very much enlarged.

An abdominal incision showed that the enlarged liver was thickly studded with small abscesses about the size of a pea.

February 23 she died.

THE FATTY LIVER.

Lesions.—There is a deposit of fat in the liver-cells, with an increase in the size of the liver. There are no changes in the stroma or in the blood-vessels. The large bile-ducts may be the seat of acute, or of chronic, catarrhal inflammation.

Causes.—The disease occurs at all ages. It is more common in women than in men. It seems to be caused by alcoholism, overeating, want of exercise, and by phthisis and other wasting diseases.

Symptoms.—1. Many fatty livers give no symptoms at all.

2. With some fatty livers there are marked symptoms of disturbance of the functions of the liver, with the physical signs of a large liver. The patients are fat, but anæmic and feeble. Mental lassitude and depression, and abnormal sensations in different parts of the body are prominent symptoms. The bowels are constipated and the functions of the stomach are deranged. There is often added a chronic gastritis, sometimes with vomiting of blood.

The treatment of these cases is by regulation of the diet and mode of life, and the use of drugs which increase the production of bile.

3. The infiltration of the liver-cells with fat may be unusually great, and the increase in the size of the liver enormous. In these patients the first symptoms are those of disturbance of the bile-producing function of the liver and of chronic gastritis. These symptoms do not improve, but get worse. The patients become so feeble and anæmic that they are confined to bed. They become a little jaundiced. They finally die in a condition of extreme exhaustion.

4. Patients with fatty livers and chronic gastritis are particularly liable to attacks of acute gastro-duodenitis with jaundice. These attacks are severe, with a good deal of fever and delirium, and often prove fatal.

5. Patients with large fatty livers may develop an increasing and fatal jaundice without obstruction of the hepatic or common bile-duct. The jaundice behaves like that which accompanies hypertrophic cirrhosis.

THROMBOSIS OF THE PORTAL VEIN.

Lesions.—The main trunk of the portal vein, or one of its branches, is partly or completely filled by a thrombus. This thrombus does not soften, is not infectious, but is replaced by new connective tissue, so that the vein is permanently obstructed.

Causes.—Cirrhosis or cancer of the liver, chronic peritonitis, tumors of the abdominal cavity, and injuries inflicted over the upper part of the abdomen act as causes. There are also cases which occur without discoverable cause, especially in old persons.

Symptoms.—First there may be the symptoms of the cirrhosis, the cancer, the chronic peritonitis, the tumors, or the injuries.

The symptoms of the thrombosis are developed rapidly. There is rapid and persistent dropsy of the peritoneal cavity. The spleen is enlarged. There are vomiting of food and of blood, diarrhœa, and bleeding from the intestines. The urine is scanty.

In some of the patients the symptoms continue without interruption. They become very feeble, delirious, and unconscious. Death takes place within a few days. In these patients there may be no dropsy. In other cases, where the dropsy is the principal feature without much bleeding or prostration, the patients may live for a long time, or even recover.

CASE XXVI. *Thrombosis of the portal vein from an injury.* Male, 14 years old, April 24.

Seven weeks ago, while at play, he was struck on the abdomen just below the ensiform cartilage by a heavy plank. He was able to get up, the skin was not broken and only a yellow mark showed the point of contusion. The pain was intense and continued so for five days, always localized in the epigastric and right hypochondriac regions.

On the fifth day his abdomen was swollen and the swelling continued to increase. At the same time he became jaundiced; this lasted for two weeks and then faded away.

For three weeks after the accident he vomited everything and had constant diarrhœa. At the end of three weeks the vomiting and diarrhœa stopped.

One week before admission he had convulsions repeated at intervals for six hours, none since.

April 24. He is emaciated, the abdomen enormously distended with fluid, the heart and lungs normal.

The abdomen was tapped and 170 ounces of clear brown serum drawn off.

After this the abdomen filled up again, temperature 98° to 103°.

June 7, a small swelling appeared near the umbilicus, became larger, broke and discharged pus.

During June this discharge continued, the abdominal swelling diminished, his general condition improved, temperature 99° to 102°.

July 10, the discharge has stopped, there is but little fluid left in the abdomen, the liver is enlarged but not the spleen.

September 10, he is apparently perfectly well.

SUPPURATIVE INFLAMMATION OF THE PORTAL VEIN.

Lesions.—The wall of the vein is thickened and infiltrated with pus. Its cavity is filled with soft, puriform, infectious thrombi. The inflammation and thrombosis extend from the trunk of the portal vein to the branches in the liver. The liver-tissue surrounding the inflamed veins also becomes the seat of suppurative inflammation. Portions of the thrombi may pass into the circulation and lodge as infectious emboli in different parts of the body.

Causes.—Fish-bones and pieces of wire have been found imbedded in the wall of the vein.

Inflammation of the umbilical vein in infants may extend to the portal vein. Abscesses in the abdominal cavity may be followed by thrombosis and inflammation, first of one of the branches of the portal vein and then of the vein itself. Calculi in the common bile-duct, which cause inflammation and ulceration of the wall of the duct, may also cause inflammation of the portal vein. There are cases in which suppurative portal phlebitis occurs without discoverable cause.

Symptoms.—The invasion of the symptoms may be sudden, or preceded by a few days of moderate fever, loss of appetite, and general malaise. Pain and tenderness over the liver are constant symptoms. They occur early in the disease and continue throughout its course.

The liver and the spleen are increased in size. Jaundice is often present. There are nausea, vomiting of food and of blood, diarrhœa, and passage of blood from the bowels.

Chills mark the onset of the disease, and are repeated throughout its course. The temperature rises at once and continues through the disease, like the temperature of pyæmia, sometimes high, sometimes low, sometimes below the normal.

There may be ascites, or local or general peritonitis.

The patients rapidly lose flesh and strength, develop cerebral symptoms, and pass into the pyæmic condition. The symptoms are evidently local—dependent on the inflammation of the vein and of the liver; and general—dependent on the septic poisoning.

The disease regularly terminates fatally at the end of about fourteen days; but it may last only a few days, or continue five or six weeks.

The most difficult cases to make out are those which occur without discoverable cause and without any marked symptoms referable to the liver. It is perfectly evident that the patients are suffering from a bad sepsis, but it is very difficult to discover the focus of infection.

CASE XXVII. *Thrombosis of the portal vein due to a fish-bone.* Male, 47 years old, February 11.

Three weeks ago, after exposure, he had symptoms of pleurisy on the right side, diarrhœa, tympanites, tenderness in the right iliac fossa, urine contained casts and albumen. He became more and more septic and died February 22.

Autopsy. The brain and heart were normal.

The left lung was adherent and there was a little fluid in the right pleural cavity.

The peritoneum contained serum, pus and fibrin.

The stomach, intestines and spleen were normal.

The liver weighed 6½ pounds. It was studded with small abscesses. The left division of the portal vein contained a firm, red and white thrombus over an inch long completely filling the vein. The right branch of the portal vein was lined with fibrin and its wall was thickened. The branches of the portal vein within the liver contained puriform fluid. There was a fishbone two inches long partly in the mesenteric and partly in the portal vein. The centre of the bone was covered by a thrombus.

CASE XXVIII. *Thrombosis of the portal vein, secondary to appendicitis.* Male, 35 years old, August 5.

In the previous December, while in St. Domingo, he had a fever which lasted for six weeks.

Four months ago he vomited blood.

August 5 he was attacked with vomiting, vomiting of blood, purging, cramps, repeated chills followed by fever, tenderness over the right side of the abdomen.

He continued to have fever, vomiting, constant pain over the upper part of the abdomen, looked more and more septic and died Sept. 15.

Autopsy. The brain was normal.

The heart was normal.

The lungs were congested and œdematous, there were infarctions in the left lower lobe.

There was a firm, red thrombus filling the portal vein and its large branches. There were numerous abscesses in the liver.

The stomach and intestines were normal.

There was an abscess around the vermiform appendix.

A branch of the superior mesenteric vein in the walls of this abscess contained a broken-down thrombus.

CASE XXIX. *Suppurative inflammation of the portal vein.* Male, 37 years old, February 3.

He was in good health until two months ago. Then he was seized with a chill, followed by fever and sweating, became weak, lost appetite and had pain in his abdomen. This was followed by chills every day for a week, and every other day for a second week. For the past month he has had chills followed by fever at irregular intervals, pain in the abdomen and has been confined to bed. For the past week he has six or seven loose watery stools every day, occasional vomiting, a little œdema of the feet

Feb. 3, pulse 118, breathing 33, temperature 102°. There are no physical signs over the heart, or lungs, the liver is of normal size, the spleen somewhat enlarged, urine normal.

He went on with an irregular septic fever, rapid loss of flesh and strength and died February 16.

Autopsy. The brain was not examined.

The heart and lungs were normal.

The peritoneal cavity contained purulent serum with a little fibrin on the peritoneum.

The small intestine was somewhat congested.

The large intestine was normal.

The liver was of medium size and pale.

The large branches of the portal vein within the liver, the main trunk of the vein and the small branches going to the small intestine and transverse colon were full of thick, purulent matter. The walls of the veins were thickened and coated with dirty fibrin.

The stomach was coated with mucus.

The spleen was somewhat swollen.

The kidneys were pale.

ACUTE INFLAMMATION OF THE GALL-BLADDER.

Most inflammations of the gall-bladder are caused by calculi, or the typhoid bacillus. But in addition there are cases which seem to be examples of a localized septic infection (Richardson, Am. J. Med. Sci., June, 1898).

Lesions.—The gall-bladder is distended and contains bile, mucus, blood and pus. The mucous membrane is congested, hæmorrhagic, or gangrenous. Its outer surface may be coated with fibrin.

Symptoms.—The symptoms are those of a confined abscess at the border of the right costal cartilages near the tip of the tenth rib. But they may resemble those of an appendicitis situated near the liver; those of an acute intestinal obstruction; those of suppuration of the kidney, or of acute pancreatitis.

Severe paroxysmal pain is regularly present. It is referred to the region of the gall-bladder, or of the epigastrium, or of the appendix, or it may be at quite a distance from the liver. The constitutional symptoms are those of sepsis: nausea, vomiting, a rapid pulse, an irregular temperature, marked prostration.

There may be a complete stoppage of gas and fæces, as if from intestinal obstruction.

These cases are often easy of diagnosis, but sometimes very difficult. If the pain and tenderness over the gall-bladder are well localized, and the distended gall-bladder can be felt it is easy, but the symptoms of sepsis with diffuse abdominal pain are more difficult.

Treatment.—Theoretically in all these cases the gall-bladder should be opened and drained. I have seen very good recoveries without operation by the continuous application of cold.

BILIARY CALCULI.

The solid portions of the bile may become separated from the fluid portions and form semi-solid, or solid, masses within the biliary passages.

1. Inspissated bile occurs in the form of soft, brown, or greenish masses, composed principally of bile-pigment, with a little cholesterin, the salts of iron, soda, and potash, and sometimes thick, tenacious mucus. While in the biliary passages it retains its viscid consistence, but when dry changes into a fine powder.

2. Biliary calculi are solid bodies composed principally of cholesterin with more or less bile-pigment. They vary as to their size, shape, color, consistence, and number.

Causes.—The normal reaction of the bile is neutral or slightly alkaline, if it becomes acid cholesterin is precipitated.

There may be such a large production of cholesterin that it cannot remain in solution.

Any conditions which retard the flow of bile into the intestine, or which render the walls of the bile-passages unhealthy, are liable to produce calculi.

Calculi are rare in chidren, but of frequent occurrence in persons over twenty years of age. They are more common in women than in men. A sedentary mode of life, constipation, excessive eating of fats, starches, and sugars, and chronic malarial poisoning, seem to have some effect in causing the formation of calculi.

I. CALCULI WHICH ARE FORMED IN THE GALL-BLADDER AND REMAIN THERE.

1. When calculi are formed in the gall-bladder, they may remain there and give absolutely no symptoms. The gall-bladder usually becomes contracted, and the cystic duct is obliterated.

2. Calculi in the gall-bladder may from time to time cause attacks of pain which last for hours or days. In some persons the attacks of pain are repeated frequently and there is much loss of flesh and strength. These cases are very difficult of diagnosis. It is of course always easy to open the abdomen and examine the gall-bladder. But the physician who has to make his diagnosis with his head rather than with his hands finds the large group of patients with attacks of the pain in the upper right side of the abdomen call for his best judgment. Many of them are permanently cured without operation and then the diagnosis remains doubtful.

CASE XXX. *Severe and long continued pain over the gall-bladder; complete recovery without operation.* Male, 24 years old.

In April, 1886, in the night, he had a sudden attack or severe pain in the right back and extending forwards, which lasted for an hour.

Three days later he had a similar attack. After that he was perfectly well until July 6, then he went to bed feeling perfectly well. Early in the morning he woke up with severe pain in the back extending forward on the right side under the ribs. After this the attacks of pain returned every night at the same time, while there was no pain in the day time.

The pain became more severe and more continuous until by September he had pain nearly all the time day and night. He was treated for gall-stones with purgatives, olive oil, electricity, opium, etc.

I first saw him on Sept. 21. He was then pale, feeble and emaciated with the face of a person worn out with pain.

It seemed to me probable that the pain at first had been due partly to intestinal colic and partly to gall-stones, but that later the pain had become largely a habit.

He was put on a milk diet and given ipecac and belladonna.

Sept. 24, the pain was no better, the same treatment was continued and a nitric acid issue made over the region of the gall-bladder.

October 1, the pain was somewhat better.

Oct. 8, the pain was as bad as ever, he was given asafœtida instead of the ipecac and belladonna.

Oct. 11, he was no better; I began to wash out the stomach every day.

Oct. 13. He was a little better, to resume the ipecac and belladonna.

Oct. 18. He was now really better, to wash out the stomach every other day.

Nov. 4, he now seemed to be well, had no more pain.

Nov. 9. He was eating three good meals a day, had gained flesh and strength, went back to work.

3. Calculi remaining in the gall-bladder may act as irritating bodies, and excite catarrhal or suppurative inflammation of the wall of the gall-bladder.

A catarrhal inflammation is attended with dilatation of the gall-bladder and pain.

A suppurative inflammation is attended with pain, dilatation of the gall-bladder, fever, sweating, loss of flesh and strength. There may be added a localized peritonitis, with the formation of adhesions.

If the patients' general condition remains good, they may be kept in bed, with the application of continuous cold over the gall-bladder. If the cases are at all threatening, the only satisfactory treatment is to open the gall-bladder and remove the calculi.

II. CALCULI MAY PASS INTO THE CYSTIC DUCT AND BECOME IMPACTED THERE.

1. There will be an attack of biliary colic, or there are several such attacks. After the last attack there will be found a tumor formed by the dilated gall-bladder. This tumor may cause so little inconvenience that no treatment for it is necessary, or it may give so much discomfort that it will be proper to empty it by the aspirator.

2. There will be an attack of biliary colic and, after this, first dilatation and then inflammation of the gall-bladder. The gall-bladder forms a tumor of large size; fever, sweating, loss of flesh and strength are developed; localized peritonitis may be added. If not treated, these symptoms will continue and cause the death of the patient.

Some of these cases may be cured by the continuous application of cold, others by aspiration, while in others it is necessary to open the gall-bladder.

3. Such a calculus may be followed by adhesions between the wall of the bile-duct and the intestine, ulceration, the escape of the calculus into the intestine, and the recovery of the patient. Or the calculus may remain in the intestine and cause an obstruction there.

4. One or more calculi may remain in the cystic duct for years and produce attacks of pain like those due to calculi in the gall-bladder. These cases have been successfully treated by surgical operations.

III. CALCULI MAY PASS FROM THE GALL-BLADDER THROUGH THE DUCTS INTO THE INTESTINE.

Such a passage of a calculus is attended with symptoms which are called an attack of biliary colic.

The cases vary as to the size and consistence of the calculi, as to whether they are true calculi or masses of inspissated bile, as to the rapidity of the passage of the calculi, and as to the frequency of the attacks.

Symptoms.—Sometimes there is an initial chill, more or less marked; but this is not constant. Then comes the most marked symptom—pain. This usually comes on suddenly and severely, referred to the epigastrium and the lower edge of the liver, and radiating in different directions. Occasionally the invasion is slower, and the pain more dull and aching. The pain, as such, resembles that of intestinal colic, and that of nephritic colic.

Nausea and vomiting frequently accompany the attack.

Fever occurs in three ways: It may simply accompany the attack. It may be due to a complicating, localized peritonitis. It may be due to pre-existing malarial poisoning.

During an attack of biliary colic, the pulse is usually small and feeble; the bowels are constipated before and during the attack; the intestines are distended with gas.

Usually the general condition of the patient remains good, but there may be marked prostration and even collapse and death.

Jaundice is regularly developed within twenty-four hours after the commencement of the pain, and continues for some days after this has subsided. But when the calculi are small, or inspissated bile is passed, the jaundice may be entirely absent. In patients who have repeated attacks of severe pain referred to the upper part of the abdomen without jaundice, it is often very difficult to tell whether the pain belongs to the bile-duct, the colon, or the stomach.

The liver may be swollen and tender during and after the paroxysm. The gall-bladder may be swollen, tender, and sometimes surrounded by a localized peritonitis.

Duration.—The ordinary attacks only last for a few hours or days. There may be only one or two attacks, or the attacks are repeated at intervals for a number of years. With such a repetition of the attack the general health may suffer, partly from the recurrence of the pain, partly from the drugs used to control it.

Less frequently there are attacks which last for weeks or months. The pain, jaundice, and vomiting continue, there is an irregular febrile movement, and the patients lose flesh and strength.

It is the fashion just now to ascribe these attacks not to the passage of the calculi but to intercurrent attacks of inflammation of the biliary passages. I think the older explanation holds good.

Diagnosis.—The regular attacks with pain and jaundice are easily recognized.

Repeated attacks of pain in the upper part of the abdomen may be due to the passage of a calculus through the ducts, to the presence of calculi in the ducts or in the gall-bladder, and to disorders of the colon or of the stomach; it is often very difficult to distinguish between them.

Treatment.—During the paroxysm of biliary colic the pain is to be relieved by hypodermic injections of morphine, or by inhalations of chloroform or ether. There seems also to be some advantage in the use of hot baths, hot-water enemata, and purgatives.

To prevent the recurrence of attacks of biliary colic there are plans of treatment which are not very rational, but yet which are often efficacious.

In some patients the attacks will cease if the stomach is washed out every day.

In some patients the use of olive-oil stops the attacks. Half a pint can be poured into the stomach through the stomach-tube; or a tablespoonful of oil can be mixed with milk and swallowed, the second day two tablespoonfuls of oil are taken, and so on up to six tablespoonfuls.

If the attacks are frequently repeated and cannot be otherwise relieved a surgical operation is necessary.

IV. CALCULI MAY PASS FROM THE GALL-BLADDER INTO THE COMMON DUCT AND BECOME IMPACTED.

The patients give the history of one or more attacks of biliary colic, or that of several attacks of pain and light-colored fæces without jaundice. Then there comes an attack of biliary colic which lasts for a time and subsides, but the jaundice continues and increases.

There are cases, however, in which there is no attack of pain to mark the time of the lodgment of the calculus. On some particular day it is evident that the common bile-duct is obstructed, and the symptoms of this obstruction become more and more decided.

The skin becomes very yellow, the urine is dark with bile-pigment, the fæces are white. There is an irregular fever, the patients lose flesh and strength; there are loss of appetite, nausea and vomiting; the bowels are constipated, the tongue is brown and dry, cerebral symptoms are developed, and there are bleedings from the mucous membranes and into the skin. The liver may be large and tender and the gall-bladder distended.

Unless relieved by a surgical operation this condition regularly proves fatal, but it is possible for the calculus to escape by a perforation into the intestine, and for the patient to recover.

The only treatment is a surgical operation to remove the calculus.

V. CALCULI MAY PASS FROM THE GALL-BLADDER INTO THE COMMON DUCT AND REMAIN THERE WITHOUT BEING IMPACTED.

Such a calculus passes into the common duct and remains there. Then for years the calculus slowly increases in size, and the common and hepatic ducts become dilated to make room for it.

The patients suffer from repeated attacks of pain, fever, nausea, vomiting, and jaundice. These attacks become more frequent and the patients cease to improve between the attacks. The jaundice becomes continuous, they lose flesh and strength, and finally die exhausted, or with suppurative inflammation of the gall-bladder or bile-duct.

The only efficient treatment is by a surgical operation.

CASE XXXI. *Movable calculus in the common bile-duct.* Male, 62 years old, March 8.

Fifteen years ago he began to have attacks of pain over the free border of the ribs on the right side, coming on suddenly, lasting from half an hour to several days and then going off suddenly. During the attacks there was vomiting, jaundice, and fever. The attacks have been repeated ever since at intervals of about six weeks. He is now in one of his attacks with temperature 103°, jaundice, pain over the liver. The liver was enlarged. His general condition was very bad, he grew worse and died on April 15.

Autopsy. The body was emaciated and jaundiced.

The right auricle and right ventricle of the heart and the pulmonary artery were dilated.

The lungs were emphysematous with old fibrous tissue and cheesy nodules at the apices.

The liver was large and hard. There was pus and fibrin on its convexity. The common and hepatic ducts were much dilated and contained a movable calculus about one inch long. The duct narrowed abruptly to its normal size as it entered the wall of the duodenum. The walls of the dilated ducts were not thickened or inflamed. The gall-stone acted as a ball valve.

The large and small bile-ducts throughout the liver were dilated, around the smaller ones were zones of connective tissue infiltrated with pus. The gall-bladder was not changed.

The spleen was normal.

The mucous membrane of the stomach was coated with mucus.

The intestines were normal.

The kidneys showed the lesions of chronic nephritis.

VI. CALCULI IN THE INTESTINES.

A calculus of some size, situated in the gall-bladder, may cause inflammation of the wall of the gall-bladder, adhesions to the intestine, perforation, and escape of the calculus into the intestine.

These morbid processes are naturally attended with symptoms, but it sometimes happens that the patient will give no history of them.

After a time the calculus is increased in size by the deposition about it of successive layers of mucus and of the salts of lime.

Such a calculus may remain in the intestine for a long time without any symptoms, except occasional uncomfortable feelings in the abdomen. It may produce attacks of severe pain. It may cause partial or complete obstruction of the intestine.

The only treatment is the removal of the calculus by a surgical operation.

Suppurative inflammation of the bile-ducts within the liver.

In many parts of the body suppurative inflammations with streptococcus infection occur without discoverable cause. In all the patients the symptoms of constitutional infection—chills, fever and emaciation are constant, the local symptoms of the inflammation are not constant. So with these inflammations of the bile-ducts within the liver. The patients are always septic, evidences of inflammation of the liver may be present or absent.

CASE XXXII. *Suppurative inflammation of the bile-ducts within the liver.* Female, 35 years old. August 2.

About eight weeks ago she was thrown against a piece of furniture and struck on the lower part of the abdomen. Soon after this she discharged a large quantity of blood from the vagina, was in bed four weeks and then partly recovered. Five weeks ago she began to have a watery diarrhœa, four to five stools daily.

Two weeks ago he had a severe chill, followed by fever, sweating, vomiting and jaundice.

She remained in this condition until she died on September 2.

Autopsy. The brain was not examined.

The heart was soft and flabby.

The lungs were œdematous. The right lower lobe was adherent to the diaphragm and invaded by an abscess of the liver which has perforated the diaphragm.

The spleen was soft and congested.

The kidneys were large and succulent.

tritis and functional disturbance of the liver. They slowly lose flesh and strength and become anæmic. It becomes more and more evident that they have a malignant new-growth in some part of the body, but it impossible to locate it on account of the want of local symptoms or physical signs.

3. There are rare cases, with a large nodular liver, which behave like cirrhosis, rather than like cancer.

4. There are cases in which the new-growth very soon invades the branches of the portal veins, so that ascites is an early and prominent symptom before there has been much change in the general health. The ascites may be accompanied [illegible] chronic peritonitis. The cases look more like chronic or [illegible] lar peritonitis, or cancer of the peritoneum, than [illegible] cancer of the liver.

Case XXXIV. *Primary carcinoma of the* [illegible] years old. February 21, 1885.

Since December, 1884, he has been [illegible] and strength with no symptom but a loss [illegible]

For two weeks past he has failed [illegible] ing, constipation and pain over the liver.

Now, he is feeble, anæmic, emaciated, [illegible] pulse 116, temperature 99°, urine [illegible] liver is much enlarged. He died in March [illegible]

Autopsy. There were a few small [illegible] monary pleura. The bronchial and [illegible] large and infiltrated with new-growth.

There was a little old thickening [illegible] valves.

The small and large intestines [illegible]

The mucous membrane [illegible] coated with mucus.

The spleen was large [illegible]

The pancreas was normal.

The kidneys were large [illegible]

The liver weighed [illegible] small white nodules. [illegible] stroma enclosing cavities [illegible] polygonal cells, others [illegible] meshes.

the liver attains a large size and gives more marked symptoms than does the primary tumor from which it originates.

Of the primary new-growths of the liver the larger number follow the type of carcinoma. They are composed of tubules lined with cylindrical epithelium, or filled with polygonal cells; or of larger and more irregular spaces packed with polygonal cells.

Primary sarcomata of the liver are of rare occurrence. They follow the type of the sarcomata which seems to be developed from the connective tissue around blood-vessels—angiosarcoma.

The new-growth may take the form of multiple nodules, of single tumors which may reach a large size, or of a diffuse infiltration. The liver itself is usually increased in size, smooth or nodular, according to the arrangement of the new-growth. But some of the livers are not enlarged, some are even diminished in size. The new-growth may compress the bile-ducts, it may compress, or grow into, the veins.

There may be secondary tumors in the peritoneum, ascites, or peritonitis.

The symptoms of the primary new-growth in the liver are variable and often obscure.

1. The patients first complain of derangements of digestion, of gastric dyspepsia, and of functional disturbances of the liver. It often happens that these symptoms follow symptoms of the same kind which existed before the formation of the new-growth in the liver, so that a history of similar symptoms may be given which extends over many years. This means that the patient first suffered from chronic gastritis and functional disturbance of the liver, and then a new-growth in the liver was formed, and the old symptoms were made worse without changing their character. As the tumor of the liver increases in size the patients grow worse. They lose flesh and strength, become anæmic, have an irregular fever, become a little jaundiced, have ascites, œdema of the legs, and a large liver. The progress of the disease is at first slow and gradual, toward the end much more rapid.

2. The patients suffer from the symptoms of chronic gas-

tritis and functional disturbance of the liver. They slowly lose flesh and strength and become anæmic. It becomes more and more evident that they have a malignant new-growth in some part of the body, but it impossible to locate it on account of the want of local symptoms or physical signs.

3. There are rare cases, with a large nodular liver, which behave like cirrhosis, rather than like cancer.

4. There are cases in which the new-growth very soon invades the branches of the portal veins, so that ascites is an early and prominent symptom before there has been much change in the general health. The ascites may be accompanied by chronic peritonitis. The cases look more like chronic or tubercular peritonitis, or cancer of the peritoneum, than they do like cancer of the liver.

CASE XXXIV. *Primary carcinoma of the liver.* Male, 47 years old, February 21, 1885.

Since December, 1884, he has been gradually losing flesh and strength with no symptom but a loss of appetite.

For two weeks past he has failed more rapidly with vomiting, constipation and pain over the liver.

Now, he is feeble, anæmic, emaciated, slightly jaundiced, pulse 116, temperature 99°, urine contains no albumen. The liver is much enlarged. He died on March 1.

Autopsy. There were a few small white nodules in the pulmonary pleura. The bronchial and mediastinal glands were large and infiltrated with new-growth.

There was a little old thickening of the aortic and mitral valves.

The small and large intestine were normal.

The mucous membrane of the stomach was congested and coated with mucus.

The spleen was large.

The pancreas was normal.

The kidneys were large and congested.

The liver weighed ten pounds. It was thickly studded with small white nodules. These were composed of a dense fibrous stroma enclosing cavities. Some of these are filled with small polygonal cells, others with a fine network enclosing cells in its meshes.

CASE XXXV. *Primary carcinoma of the liver, clinical history of cirrhosis.* Female, 55 years old.

December 25, 1886, she began to have some pain and swelling of the abdomen.

In February, 1887, her feet began to swell.

March 15, she has marked ascites, dropsy of the legs, pulse 88, temperature 98°, urine 1.020, trace of albumen.

The liver is enlarged downward.

March 18, she is out of bed, 14 ounces of urine in twenty-four hours. She continued to look as if she had cirrhosis of the liver with dropsy, lost flesh and strength and died April 7.

Autopsy. Both pleural cavities were half full of serum.

There was a little old thickening of the aortic and mitral valves of the heart.

The peritoneal cavity was distended with serum.

The spleen was enlarged and studded with small, white nodules.

The stomach was coated with mucus.

The small and large intestine were normal.

The pancreas, kidneys, uterus and ovaries were normal.

The liver weighed seven pounds. The right lobe was thickly studded with large and small white nodules. These were composed of a fibrous stroma enclosing regular tubular spaces filled with polygonal nucleated cells.

CASE XXXVI. *Cysts of the liver and kidneys.* Male, 37 years old, July 6.

For six months he has had dyspnœa on exertion, general dropsy, headache, and vomiting.

Now, he is emaciated, no dropsy, dyspnœa, pulse 84, temperature 99°, urine 1.003, albumen, no casts, the liver is enlarged.

He became comatose and died July 13.

Autopsy. The brain was normal.

The aortic and mitral valves were thickened, the left ventricle was hypertrophied.

The lungs were very œdematous.

The liver was large, it contained numerous cysts from the size of a filbert to that of a lemon, they had smooth walls and contained yellow serum.

The spleen was normal.

The mucous membrane of the stomach was coated with mucus.

The kidneys were very large and changed into a mass of cysts.

Cancer of the Common Bile-Duct.

Lesions.—The growth begins in the wall of the common bile-duct near the duodenum and surrounds and constricts the duct. The growth may remain small, or may extend to the pancreas and lymphatic glands and form large tumors. The growth constricts the bile-duct, prevents the escape of bile, and so produces dilatation of the bile-ducts and the gall-bladder.

The disease belongs to persons over forty years of age.

Symptoms.—Jaundice is developed suddenly, and increases in intensity throughout the disease.

Pain comes on suddenly, and is referred to the region of the common bile-duct.

Nausea and vomiting often mark the onset of the symptoms and may continue.

The sudden development of the symptoms in a disease of this character is a noticeable feature.

The patients go on with the symptoms of a very bad obstructive jaundice. The pigmentation of the skin becomes constantly deeper, the urine is loaded with bile-pigment, the fæces are white. The patients lose flesh and strength, there are hemorrhages in the skin and from the mucous membranes, cerebral symptoms are developed, the tongue is brown and dry, the gall-bladder may be dilated so as to form a tumor of some size. The patients finally die deeply jaundiced, emaciated, and semi-comatose.

The duration of the symptoms is usually for from one to three months.

Cancer of the Gall-Bladder.

Lesions.—The growth is said to begin in the wall of the gall-bladder near the fundus or the cystic duct, and then extends until it involves a considerable part of the bladder. The gall-bladder may become contracted and small, or it may contain gall-stones, or it may be distended with fluid so as to form a tumor of some size. There may be secondary nodules in the liver.

Symptoms.—The clinical histories are very obscure. The patients lose flesh and strength and become cachectic, as if they had a cancer in some part of the body.

If the gall-bladder is contracted there is nothing to tell us where the cancer is.

If the gall-bladder is dilated, the presence of the tumor assists in making the diagnosis.

If the liver is enlarged and contains secondary nodules, the case is usually supposed to be one of cancer of the liver.

CASE XXXVII. *Carcinoma of common bile-duct.* Male. 48 years old, November 9.

Three weeks ago he was suddenly attacked with jaundice, nausea, vomiting and prostration.

Nov. 9, he is emaciated, deeply jaundiced, tongue brown and dry, pulse 80, temperature 96°, urine contains bile-pigment. There is a continual bloody discharge from the mouth.

Nov. 11, the distended gall-bladder can be felt from the eleventh rib to the anterior superior spine of the ileum. I aspirated sixteen ounces of bile.

Nov. 13, not as yellow, aspirated eight ounces of bile.

November 16, died.

Autopsy. Body emaciated and deeply jaundiced.

The brain was not examined.

The lower lobe of the right lung was hepatized.

The aortic and mitral valves were a little thickened.

The spleen was normal.

The capsules of the kidneys were adherent, the capsule thinned.

The stomach, small intestine and large intestine were normal.

The liver was of medium size and bile stained.

The gall-bladder was distended with bile. The cystic, hepatic and large ducts in the liver were moderately dilated. Where the common duct passed over the head of the pancreas its wall was thickened and its lumen nearly obliterated.

The pancreas was small, hard and nodular.

The bile-duct, where it passed over the pancreas, had its wall thickened with dense connective tissue in which were irregular tubules lined with cylindrical epithelium. Beneath this in the pancreas was a mass of connective tissue in which were large tubules lined with cylindrical epithelium. Through the rest of the pancreas there was a diffuse growth of connective tissue, atrophy of the glandular acini and scattered tubules lined with cylindrical epithelium.

CASE XXXVIII. *Carcinoma of the common bile-duct.* Female, 35 years old, April 30.

Two months ago, after exposure to cold, she was seized with severe pain in the epigastrium and became intensely jaundiced. The pain continued for four weeks. She is now very feeble and deeply jaundiced.

May 28 she began to vomit blood and continued to do so until she died on May 30.

Autopsy. On the inner surface of the dura mater over the convexity of the brain there was a thin layer of new tissue in which were thin clots. The serum beneath the pia mater was increased in quantity.

The heart was normal.

The bronchi contained blood clots.

The liver was large, greenish, adherent to the stomach. At its left border was a small abscess between the liver, spleen and diaphragm.

The common, cystic and hepatic ducts were dilated. The termination of the common duct in the duodenum had its wall much thickened and projected into the duodenum like a teat. It was not occluded, but admitted a probe as large as a goose quill. The bile-ducts throughout the liver were dilated and contained pus.

The thickening of the wall of the common bile-duct was due to a new growth composed of follicles lined with cylindrical epithelium. Over the new growth the mucous membrane was intact. The new growth invaded a small part of the adjacent pancreas.

The stomach was coated with mucus and contained blackish fluid.

CASE XXXIX. *Carcinoma of the common bile-duct confined to its intestinal portion.* Male, 78 years old.

He was a clergyman and had suffered from dyspepsia for fifty years. December, 1885, he had a severe attack of pain in the epigastrium and vomiting.

January, 1886, he had a similar attack. From these attacks he rallied slowly and imperfectly. His stools became lighter colored and there was occasional diarrhœa. He gave up one article of food after another, because they caused pain and vomiting.

In June, he continued to lose flesh and there was general itching of the skin.

In July he became decidedly jaundiced with clay colored stools, constant nausea and attacks of pain.

Oct. 11 I saw him. He was then feeble and emaciated slightly jaundiced, several loose white stools every day, no abdominal tumor. I thought he had a permanent obstruction of the common duct, probably from a calculus. He gradually failed and died November 1.

Partial Autopsy. The liver was large and bile stained. The gall-bladder contained eight ounces of turbid fluid. The common and hepatic ducts were dilated with thickened walls up to the duodenum. The papilla of the common bile-duct in the duodenum was changed into a tumor three-quarters of an inch long and one-quarter of an inch thick. It was composed of a connective-tissue stroma enclosing tubular follicles lined with cylindrical epithelium.

CASE XL. *Carcinoma of the common bile-duct.* Male, 55 years old, February 28.

Two months ago he began to have jaundice, vomiting, pain over the liver and gradual loss of flesh and strength.

He continued after this to get steadily worse, increasing jaundice, emaciation, vomiting, stupor. The distended gall-bladder could be easily felt. April 12 he died.

Autopsy. The body was emaciated and deeply jaundiced.

The heart was normal.

The lungs were somewhat congested, the bronchi congested and coated with mucus.

The kidneys showed the lesions of chronic nephritis.

The mucous membrane of the stomach was blackish and thickly coated with mucus.

The liver was large and green, the larger bile-ducts distended with bile. There were a number of small white nodules in the right lobe. The gall-bladder was dilated and distended with bile.

The end of the common bile-duct in the duodenum was enlarged so as to project like a teat. Its wall was thickened by a growth of connective tissue containing tubular follicles and its lumen was filled by an outgrowth of similar tissue. Further back in the head of the pancreas the walls of the bile-duct and pancreatic duct were thickened with the same tissue. In the surrounding pancreas was a growth of connective tissue and tubules.

The nodules in the liver were composed of tubular and irregular follicles lined and filled with cylindrical cells.

CASE XLI. *Carcinoma of the common bile-duct.* Male, 47 years old, June 1.

Fifteen months ago he began to have dysentery, from which he has never recovered.

Twelve months ago he had two attacks of hæmatemesis. He has every day three or four passages of blood and mucus.

Five days ago he had headache, loss of appetite and became jaundiced.

June 1, he is feeble, jaundiced, with nausea, pain in the left side, urine contains a little albumen.

June 31, the jaundice has been increasing, he is losing flesh and strength, continues to pass blood and mucus from the bowels. He went on in this way and died August 4.

Autopsy. The body was deeply jaundiced with small ecchymoses in the skin.

The brain was not examined.

The heart was normal.

There were old pleuritic adhesions, emphysema, old fibrous nodules and diffuse fibrous tissue at both apices.

The spleen weighed 1½ pounds.

In the pancreas the small ducts were dilated.

The stomach was distended with blood, no ulcers.

The small intestine contained recent blood.

The colon was studded with old ulcers.

The liver weighed four pounds, it was yellow, the hepatic and cystic duct and gall-bladder dilated. Around the common bile-duct for one and a half inches up from the duodenum was a tumor the size of a pigeon's egg constricting the common duct.

In the liver there was a small growth of connective tissue along the veins and bile-ducts. The portions of the tumor near the bile-duct and duodenum were of the type of cylindrical celled carcinoma.

Case I.—Catarrhal jaundice with high temperatures.

Case II.—Catarrhal jaundice with diminution in the size of the liver.

Case III.—Chronic gastro-duodenitis with jaundice, improvement.

Case IV.—Fatal jaundice caused by chronic duodenitis.

Case V.—Acute yellow atrophy of the liver.

Case VI.—Acute yellow atrophy of the liver.

Case VII.—Acute yellow atrophy of the liver.

Case VIII.—Acute yellow atrophy of the liver.

Case IX.—Acute yellow atrophy of the liver.

Case X.—Acute yellow atrophy of the liver.

Case XI.—Cirrhosis, small smooth liver, loss of nutrition and dropsy.

Case XII.—Cirrhosis, large smooth liver, loss of nutrition, dropsy and jaundice.

Case XIII.—Cirrhosis, hæmatemesis.

Case XIV.—Cirrhosis, chronic peritonitis.

Case XV.—Cirrhosis, fatal jaundice.

Case XVI.—Cirrhosis, fatal jaundice.

Case XVII.—Thickening of the capsule of the liver with dropsy and vomiting of blood.

Case XVIII.—Abscess of the liver, aspiration, recovery.

Case XIX.—Abscess of the liver, aspiration, recovery.

Case XX.—Abscess of the liver, death.

Case XXI.—Abscess of the liver, incision, recovery.

Case XXII.—Abscess of the liver, rupture through the diaphragm.

Case XXIII.—Abscess of the liver, secondary pericarditis.

Case XXIV.—Abscess of the liver, perforation of the pleura and peritoneum.

Case XXV.—Small multiple abscesses of the liver.

Case XXVI.—Thrombosis of the portal vein from an injury.

Case XXVII.—Thrombosis of the portal vein due to a fishbone.

Case XXVIII.—Thrombosis of the portal vein secondary to appendicitis.

Case XXIX.—Suppurative inflammation of the portal vein.

Case XXX.—Pain over the gall-bladder, recovery.

Case XXXI.—Movable calculus in the common bile-duct.

Case XXXII.—Suppurative inflammation of the bile-duct.

Case XXXIII.—Suppurative inflammation of the bile-duct.

Case XXXIV.—Primary carcinoma of the liver.

Case XXXV.—Primary carcinoma of the liver.

Cace XXXVI.—Cysts of the liver and kidneys.

Case XXXVII.—Carcinoma of the common bile-duct.

Case XXXVIII.—Carcinoma of the common bile-duct.

Case XXXIX.—Carcinoma of the common bile-duct.

Case XL.—Carcinoma of the common bile-duct.

Case XLI.—Carcinoma of the common bile-duct.

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